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HYPERPLASTIC CHOLECYSTOSES*

HICKEY LECTURE, 1960

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It is my great good fortune to have been asked to deliver the twenty-fourth Hickey Memorial Lecture. I am pleased and proud, and deeply grateful, to be so honored.

Dr. Preston M. Hickey died in 1930, the same year that I started my work in radiology. Fortunately, his writings in the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE *and his reputation, which lives on in the hearts of his colleagues, make me feel that, in a way, I have come to know him.*

A lasting monument to this distinguished radiologist is the continuing growth of the AMERICAN JOURNAL OF ROENTGENOLOGY, *which began as the* Quarterly of Roentgenology *of which Dr. Hickey was founder and the first Editor. Being an enthusiastic scientist and a tireless worker, he personally arranged all the details of editing, printing and the distributing of the Journal with the ability, persistence and faith of a true pioneer. Both as a practitioner and as an editor, he was in the forefront of those who were constantly striving to elevate the practice of radiology to its present high status in the field of medicine.*

I feel a special warmth for Dr. Hickey because of the personal ties he had with France and Montreal. In France during the First World War, he was Chief of the X-Ray Division of the United States Army. During this time, he counted amongst his closest friends such distinguished radiologists as Dr. Ledoux-Lebard and Dr. Antoine Béclère. When Dr. Ledoux-Lebard was invited to America in 1922 to give the Caldwell Lecture, Dr. Hickey was one of the instigators of this event. Dr. Hickey was also a personal friend of two prominent Montreal radiologists, Dr. Pirie, Professor of Radiology at the University of McGill, and Dr. Pariseau of the University of Montreal.

Dr. Hickey was a great radiologist because he had the imagination of a true pioneer. He refused to be bound by the restrictions of unperfected equipment and procedures and by the routine standards of his day. For those in our profession who feel weighted with standardized techniques and stereotyped judgments, this attitude should be the source of the greatest inspiration in the continuing effort to penetrate the perplexing problems of today. Such a perplexing problem is the domain of cholecystoses, the elucidation of which should be tempting to the inquiring mind.

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HISTOPATHOLOGISTS, no doubt from force of habit, continue to use the term "chronic cholecystitis" for a wide variety of gallbladder alterations; among these chronic cholecystopathies there are instances where an excessive proliferation of normal tissue components is the fundamental part of the process. It is our contention that these hyperplastic reactions are degenerative in nature rather than inflammatory.

We have grouped these processes under the generic title of "hyperplastic cholecystoses" to underscore not only their proliferative and degenerative character but also their tendency to appear simultaneously or consecutively in the same gallbladder and because it is our assumption that they possess a common etiology.

To be more precise, we have chosen this new term to cover several pathologic processes for three main reasons:

1. Hyperplasia implies a multiplication of tissue elements whereas an increase in their size would be hypertrophy.

2. The term "cholecystosis" denotes that the hyperplasia described may be related to inflammation only incidentally. It also stresses that this hyperplasia is a disordered result of overwork or perhaps a premature manifestation of senescence in certain tissues under the influence of endocrine or metabolic factors in relation to biologic type, age and sex. This hypothesis is supported by the fact that estrogens experimentally administered to female guinea pigs produce uterine fibromyomas and hyperplastic changes in the gallbladder quite comparable to the lesions we call cholecystoses (Tangari,[32] Moricard, Albot and Bonnet[3]).

3. The presence of one form of cholecystosis in a given gallbladder does not preclude the coexistence or eventual appearance of several other forms. On the contrary, the frequency of such associations is an indication that hyperplasia of one of the cellular components of a normal gallbladder induces excessive overproduction in other components.

Even if one considers this concept of a single etiology debatable, it is very useful in clinical roentgenology as a constant reminder to the observer of the frequent coexistence of the various lesions. The radiologist may hesitate before diagnosing on minute findings one form of cholecystosis but his opinion will be much firmer if he is able to demonstrate morphologic or functional changes of an accompanying process.

One should never lose sight of the fact that cholesterolosis, adenomyomatosis and neuromatosis frequently coexist; for this reason, when one type is discovered, an earnest search should be made for the other two. This notion expresses the synergism of overdeveloped tissues acting jointly to bring about what we call the "hyperfunction complex," that is, hyperconcentration, hyperexcitability and hyperexcretion.

The present classification excludes the contracted and thickened gallbladders of chronic cholecystitis, which are the scarred end stages of long standing inflammatory mutilations.

We thus distinguish "chronic cholecystitis," which results from a true inflammation: physical, chemical, bacterial or allergic in nature; and "hyperplastic cholecystosis," an abnormal state which is characterized by an increase of tissue elements. In such hyperplasias the multiplication of cells is orderly and does not possess the destructive capacities of neoplasia.

The objection may be raised that the gallbladders affected by one of the various types of these so-called cholecystoses, being often infiltrated by leukocytes, are actually the seat of a true inflammation. This argument does not necessarily hold true since it is an admitted fact that leukocytes and plasmocytes are often found in the normal gallbladders of elderly persons. In true inflammatory reactions leukocytes are more numerous and more densely grouped.

In a discussion on gallbaldder dysplasias Albot and his co-workers[4] (1954) grouped into a single entity several tissue processes such as adenofibromyomatosis, cholestero-

Table I

HYPERPLASTIC CHOLECYSTOSES

	Synonyms
1. *Cholesterolosis*	
(A) Diffuse type (disseminated micronodules)	strawberry gallbladder (MacCarthy[22])
	cholesterosis (Mentzer[25])
	lipoid cholecystitis (Boyd)
(B) Polypoid type	pedunculated polyp: solitary or multiple
	sessile polyp
2. *Adenomyomatosis*	
(A) Generalized type	cholecystitis glandularis proliferans (King and MacCallum[17])
(B) Segmental type	cystic cholecystitis (Bodnar[3])
	intramural diverticulosis (Rokitansky, Luschka, Aschoff)
	pneumocholecyst with intramural bullae (Åkerlund and Rudhe[2])
	parietal calculi (Chiray and Pavel[7])
(C) Localized type	adenomyoma (Sutherland,[30] Nicod[27])
	adenoma
	adenofibromyoma
	epitheliomyoma (Caroli *et al.*[6])
	myoepithelial anomaly (Jones and Walker[11])
3. *Superficial Neuromatosis* (Riopelle[28])	
4. *Deep Neuromatosis*	
5. *Elastosis* (Riopelle[28])	
6. *Lipomatosis*	
7. *Interstitial Fibromatosis*	
8. *Pericholecystic Fibromatosis*	
9. *Hyalinocalcinosis*	porcelain gallbladder
	calcified gallbladder
	calcifying cholecystitis

losis and certain strictures and kinkings. The concept that the above lesions originated from common causes had also been suggested by Savy, Bonnet and Martin in 1913 and by Caroli *et al.*[6] in 1951.

In 1942, Riopelle[28] first described neuromatosis and in 1946 elastosis, two types of hyperplasia which he often found coexisting with cholesterolosis, adenomyomatosis and other slowly progressing alterations of the gallbladder.

In our opinion, the term "hyperplastic cholecystoses" would be more appropriate than dysplasias, which refer to malformations in general without distinguishing between an embryonal anomaly, a growth defect, or a sequel to an acquired disease.

The work of Albot and his co-workers[3,4] on gallbladder dysplasias was very enlightening to us. A unitarian pathogenetic concept made possible the grouping of a variety of interrelated lesions in a specific class, separated from the true inflammatory processes. Although a new idea of chronic gallbladder conditions evolved, Albot's theory was not fully satisfactory. His list was incomplete and the term "dysplasia" could suggest a congenital anomaly. In an effort to improve Albot's unitarian concept, we enlarged the group of the various hyperplasias that seemed related to common biologic influences and selected a name that suggested both the nature and etiology of the lesions.

We will not discuss in minute detail the pathologic aspects of all the varieties of cholecystosis listed in Table 1 and the interrelationships. We shall deal specifically with cholesterolosis, adenomyomatosis and neuromatosis because of the rapidly growing interest in these conditions on the practical level, that is, in the daily use of cholecystography and, consequently, in the fields of medicine and surgery. Elastosis and hyalinocalcinosis (porcelain gallbladder) also have a theoretic and practical

relationship to the above mentioned conditions; however, it is not the purpose of this paper to discuss this aspect.

TECHNICAL CONSIDERATIONS: SELECTIVE CHOLECYSTOGRAPHY

Roentgenologic signs of hyperplastic cholecystoses can be observed on cholecystograms obtained with the simplest techniques, but the chances of accurate diagnosis increase with more elaborate procedures.

Since 1940, it has been a regular practice in our department to examine all gallbladders with a combination of fine and varied techniques which constitute what we call "selective cholecystography." During the past twenty years, exceptions to this rule have been very rare and only an occasional cholecystogram was taken by a technician. This means that each year more than 4,000 cholecystographies are carried out personally by radiologists using spot roentgenograms in upright, prone, recumbent, oblique, lateral and all other positions or angles deemed useful from the fluoroscopic observation. The degree of compression, voltage, milliamperage, time exposure and other factors are also varied according to the examiner's aim and choice. The examination is repeated after a fat meal which is very seldom omitted. As a rule, large gallbladders are re-examined within forty-five minutes after ingestion of the fat beverage; small gallbladders are re-examined within thirty minutes after the cholecystokinetic intake. Deviations from this routine are permitted only in the shortening of the time interval. Whenever the fluoroscopist feels that a drainage study may improve his final judgment or if the patient is re-examined because dubious abnormalities were shown on previous cholecystograms, the time interval between the fat meal and the beginning of a new roentgenographic series may be reduced to five minutes.

For special evacuation studies, we use the prone position and the conventional duodenal serialograph producing four different images on a 10×12 inch film. The exposures

FIG. 1. Opaque material in the duodenal loop and hepatic flexure aids in distinguishing superimposition of gas on the gallbladder shadow. The semitransparent annular rim and the opacified crater and minute diverticula at the tip of fundus of the gallbladder indicate the presence of adenomyoma.

are made rhythmically at three, four or five minute intervals depending on the problem to be elucidated and the speed of emptying which had been previously evaluated.

For the fat meal, two egg yolks mixed with two ounces of cream (Boyden's recipe) directly out of the refrigerator is the only preparation that has proved to be of real value in our experience.

The position of the gallbladder over the fenestra in the lead shield is carefully chosen under fluoroscopic control.

About two-thirds of our cholecystographies are performed conjointly with a barium enema study and/or a barium meal examination. We have noted that ice cold drinks are the best stimulants of the gallbladder motility. The opaque material in the digestive tract helps to distinguish superimposition of gas on the gallbladder shadow (Fig. 1). The emptying of the colon facilitates compression and clears the biliary reservoir of confusing shadows, thus minimizing the elements of error.

FUNCTIONAL OR INDIRECT ROENTGEN SIGNS

Since cholesterolosis is associated with catarrhal cholecystitis, lithiasis, neuromatosis, adenomyomatosis, strictures, kinkings, etc., it shares in causing functional disturbances in the gallbladder. The importance of its role as a causative factor is, however, impossible to determine.

When the opacification of the gallbladder is impeded by stones, congestion, spasm or any mechanical or functional obstacle, cholesterolosis or any of the cholecystoses need not be indicted. An unrelated disease of the digestive tract or some other organ system, an incorrectly administered contrast medium and many other factors may be the cause of poor visualization, whether the gallbladder is hyperplastic or not. We stress this fact because most early reports on the roentgenographic diagnosis of cholesterolosis stated that this condition hinders opacification. These workers did not possess our present means of investigating the state of the bile ducts or of accurately timing the administration and excretion of the contrast material. With intravenous cholangiography (biligrafin, cholografin, radio-selectan, etc.), we can determine the amount of contrast medium to be excreted by the liver and also the exact moment of administration of the medium. Moreover, in studying the excretion of the medium, intravenous cholecystocholangiography offers a marked advantage in that once the medium has reached the bowel it does not return to the biliary system, thus avoiding the successive passages of the contrast medium through the bile ducts that follow the administration of peroral media.

During 1954 and 1955 we utilized intravenous cholangiography (biligrafin) in over 200 patients in order to compare the functional behavior of normal gallbladders and of gallbladders that showed certain alterations following an orally administered contrast medium.

Our findings permitted us to confirm the statement of Kirklin, Taylor and Bollman (1927)[18] that the mucosal hyperplasia of cholesterolosis favors opacification (Table II). We were also able to better understand the individual roles played by neuromatosis and adenomyomatosis; but—of more consequence—we observed how each type of hyperplasia increased its influence on gallbladder function when it was associated with one or both of the other types of hyperplasia.

TABLE II

OPACIFICATION OF GALLBLADDER IN PROVED CASES OF CHOLESTEROLOSIS

HÔTEL-DIEU DE MONTRÉAL, YEAR 1955

	No. of Cases	Per Cent
Gallbladder not visualized	1	2.1
Gallbladder faintly opacified	3	6.5
Opacification less than skeletal opacity	5	10.9
Opacification equal to skeletal opacity	12	26.1
Opacification greater than skeletal opacity	26	54.4
	47	100

This synergistic concept helps to unravel the complex functional disturbances revealed by cholecystography and to discover the responsible anatomic lesions.

To the hyperplastic triad comprising cholesterolosis, neuromatosis and adenomyomatosis there is a corresponding hyperfunctional triad, hyperconcentration, hyperexcitability, and hyperexcretion.

HYPERCONCENTRATION

Intravenous cholangiography reveals that an excess of mucosa or muscle increases the distensibility of the gallbladder. The latter may stretch to an enormous size by a too rapid and abundant influx of the medium-containing bile. The ordinary bile flow induced by biligrafin is not always so abundant as to considerably distend the gallbladder but this capability is usually increased in the initial phase.

The gallbladder returns to normal size about two hours after the administration of the contrast medium, demonstrating the good elasticity of the wall. During this

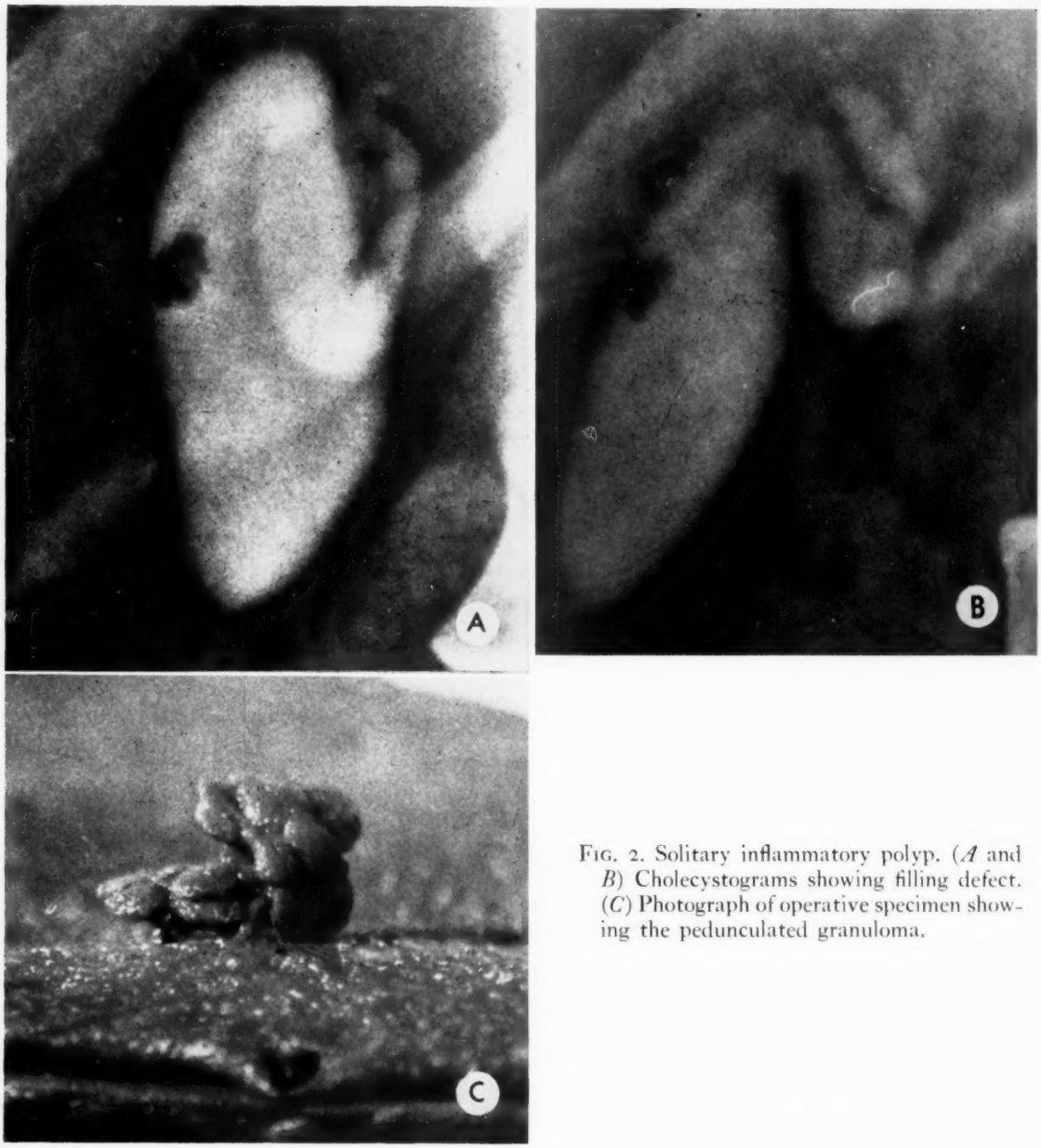

FIG. 2. Solitary inflammatory polyp. (*A* and *B*) Cholecystograms showing filling defect. (*C*) Photograph of operative specimen showing the pedunculated granuloma.

time the decrease in size is accompanied by an increase of opacification.

The intensity of concentration reaches its maximum around the third hour. From then on density and size remain unchanged until the ingestion of the fatty meal.

In a series of 47 proved cases of cholesterolosis, 26.1 per cent of the gallbladders showed a degree of opacification equal to the opacity of neighboring vertebrae, which we consider as average, but in 54.4 per cent of the cases this degree was exceeded (Table II).

HYPEREXCITABILITY

This phenomenon is observed in the short latent period between the ingestion of fatty substances and the first ejaculations of vesicular bile into the cystic and common bile ducts. It occurs so early that, in order to view it, roentgenograms must be taken

no later than five minutes after ingestion of the fatty meal.

HYPEREXCRETION

The contractile force is evaluated by the speed with which the gallbladder eliminates its content and by the degree of maximum reduction in size before the delivery of new bile from the liver. In some cases with cholecystoses, the gallbladder is completely evacuated within thirty minutes after the ingestion of fat.

In summary, in cases of mucosal hyperplasia an increase is noted in the speed and intensity of the concentration of the contrast medium; in neural hyperplasia an acceleration in the response to stimuli is observed; and in muscular hyperplasia there is an exaggerated contractile action as well as faster and more complete expulsion of the opaque bile from the gallbladder.

The annular strictures found in cholesterolosis and adenomyomatosis and ascribed to fibrosis are very often simply segmented manifestations of hyperkinesia, for they are seen to relax. This observation becomes more manifest when additional roentgenograms are made after the administration of spasmolytic drugs.

CHOLESTEROLOSIS

GENERAL CONSIDERATIONS

Lipoid corpuscles are usually seen at the base of the epithelial cells; however, the main cholesterol deposits are located in the lamina propria in overdistended histiocytes. Such fat-laden macrophages are called foam cells. As they accumulate into more voluminous collections, they raise the epithelium and give rise to typical yellowish flecks as well as sessile and pedunculated excrescences over the inner surface of the viscus. Some lipoid material may also spread freely throughout the stroma. Pathologists refer to this type of fat impregnation as xanthomatosis.

Virchow (1857), Naunyn (1892) and Aschoff (1906) described deposits of neutral fat and cholesterol in the gallbladder wall. Moynihan[26] (1909) was the first to report this condition as a clinical entity. In 1910, MacCarthy[22] coined the expression "strawberry gallbladder." In 1912, Laroche and Flandrin emphasized the frequency of stones in strawberry gallbladders. Policard (1914) and Boyd (1922) demonstrated that the substance causing the lipoid infiltration was esterified cholesterol. Lichtwitz[21] (1914), noting the fragility of the pedicle of certain cholesterol-laden warts, hypothesized that stones could result from the rupture of the stalk with consequent liberation of these small vegetations into the lumen which then become the starting points of calculi. Several terms have been proposed as synonyms for strawberry gallbladder: fish-scale gallbladder, lipoid gallbladder, lipoid cholecystitis, lipoidosis, cholesterosis, etc. Mentzer[25] feels that comparisons with strawberries, fish-scales, etc., are not appropriate to the scientific language; he maintains that cholesterolosis is the only valid name. Since pathologists actually recognize the term as being the most convenient, it seems that clinicians and radiologists should make its acceptance unanimous.

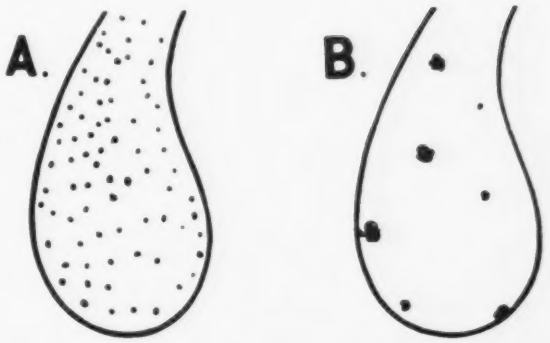

Fig. 3. Diagrams of cholesterolosis. (*A*) Diffuse granular or punctiform type. (*B*) Disseminated multipolypoid type.

Yellow or whitish cholesterol seed-like dots scattered over the mucosal membrane that has become red by intercurrent hyperemia are the reason for the comparison with a ripe strawberry (Fig. 4*C;* and 5*C*). These specks vary in size, shape, number

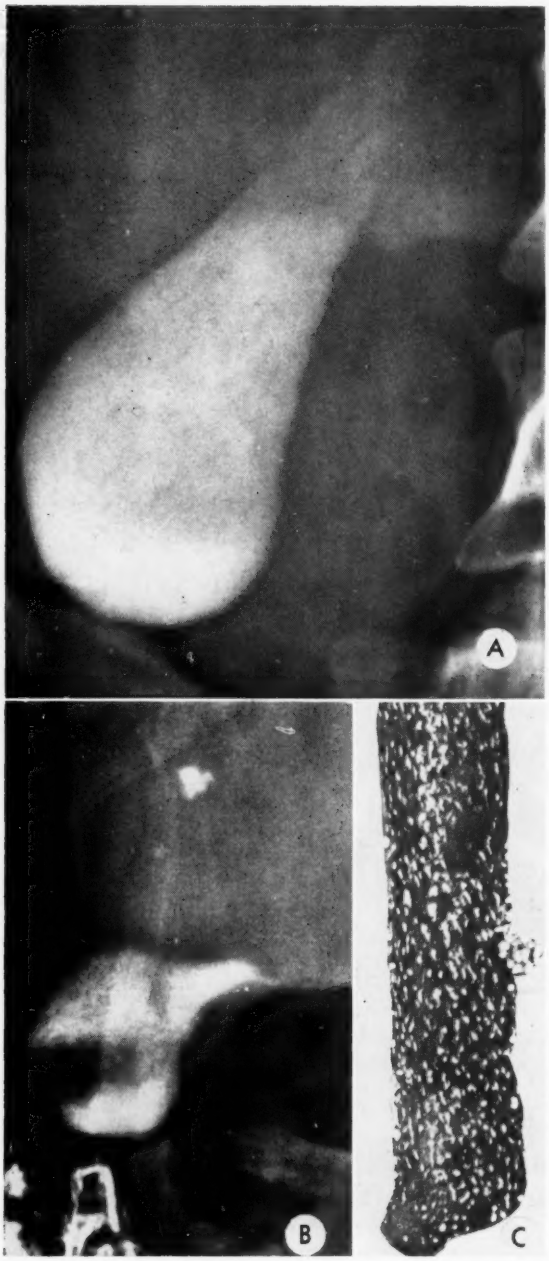

FIG. 4. (*A* and *B*) Diffuse granular or punctiform cholesterolosis. (*C*) Gallbladder everted over a test tube. Cholesterol specks and warts are seen and also a large pedunculated polyp. An adenomyoma is bulging at the tip.

and distribution. The fundamental micronodule is no larger than a millimeter. In the diffuse form, hundreds of these pearly nodules may cover a patch or the whole inner surface of the viscus.

At first, the lipoid corpuscles are rounded; later they develop into lobulated or wartlike sessile excrescences and still later they take on the shape of pedunculated polyps with a stem several millimeters long but very thin and tenuous. We have observed on two occasions a solitary cholesterol polyp become detached in the biliary sac during an operation when the surgeon attempted to feel the minute mass by pinching and rolling the walls between his fingers. Since then, we are inclined to accept Lichtwitz's[21] theory according to which cholesterol polyps may be set free and initiate the development of stones.

We have in our files several examples of a solitary cholesterol polyp. However, it must be stressed that cholesterolosis is usually characterized by numerous disseminated granules or polyps. Thus, one may distinguish the diffuse granular or punctiform type (Fig. 3*A;* and 4, *A–C*), and the disseminated multipolypoid type (Fig. 3*B;* 5, *A–C;* and 6, *A–C*). When cholecystograms demonstrate a single lacunar shadow, a differential diagnosis between a solitary cholesterol polyp and papilloma (Fig. 7*A*), adenopapilloma and inflammatory granuloma (Fig. 2, *A–C*) is practically impossible. These latter conditions, however, are even more infrequent than the single cholesterol polyp.

We disapprove the indiscriminate use of the term "papilloma" for any type of polypoid growth within the gallbladder because papillomas and adenopapillomas are the only truly neoplastic lesions with malignant propensities; also, because Tabah and McNeer[31] have shown malignant transformations in authentic papillomas and adenopapillomas. When a single lacunar shadow is observed, it should not be called a papilloma but rather a polyp of unknown etiology, although on a statistical basis a cholesterol polyp is the most probable lesion.

ROENTGENOLOGIC DIAGNOSIS

Feci and Ruggieri[9] (1931) were the first to point out that the diagnosis of cholesterolosis was feasible by means of cholecystography. However, these authors and,

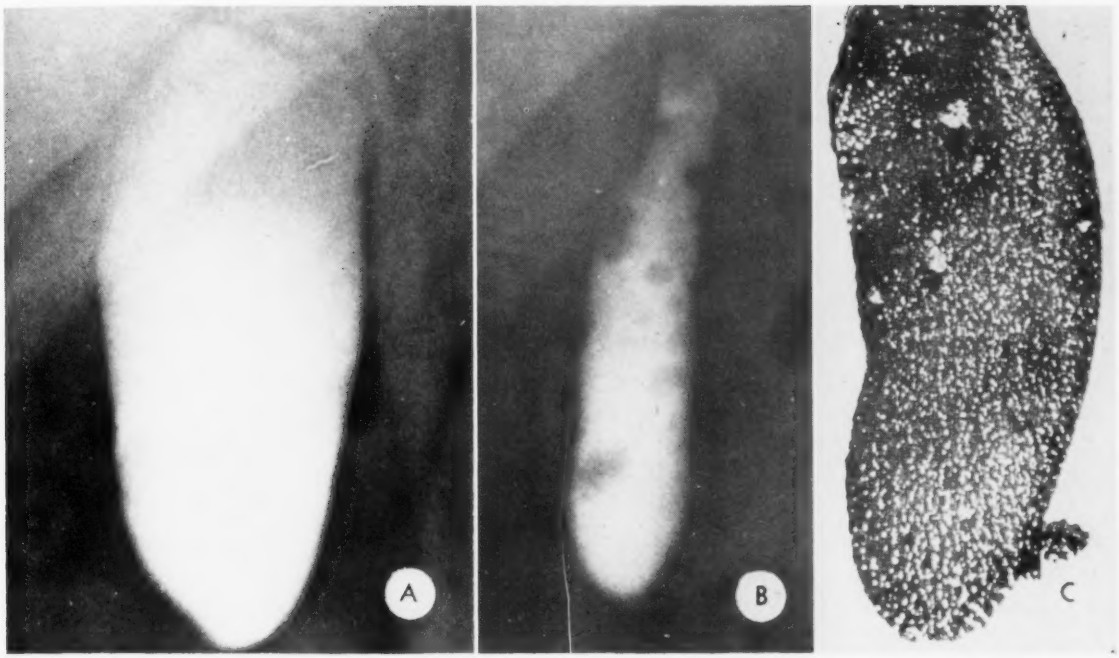

Fig. 5. (*A–C*) Diffuse punctiform and disseminated multipolypoid cholesterolosis. Hyperconcentration and rapid emptying of the gallbladder. The polyps show considerable variation in size.

later, many others based their diagnosis on functional and morphologic criteria.

It was Kirklin[18] (1931) who really paved the way to the roentgenologic diagnosis of cholesterolosis. Unfortunately, he referred to all varieties of small proliferations on the inner surface of the gallbladder as papillomas without any histologic discrimination, thus initiating a confusion in terminology which still prevails.

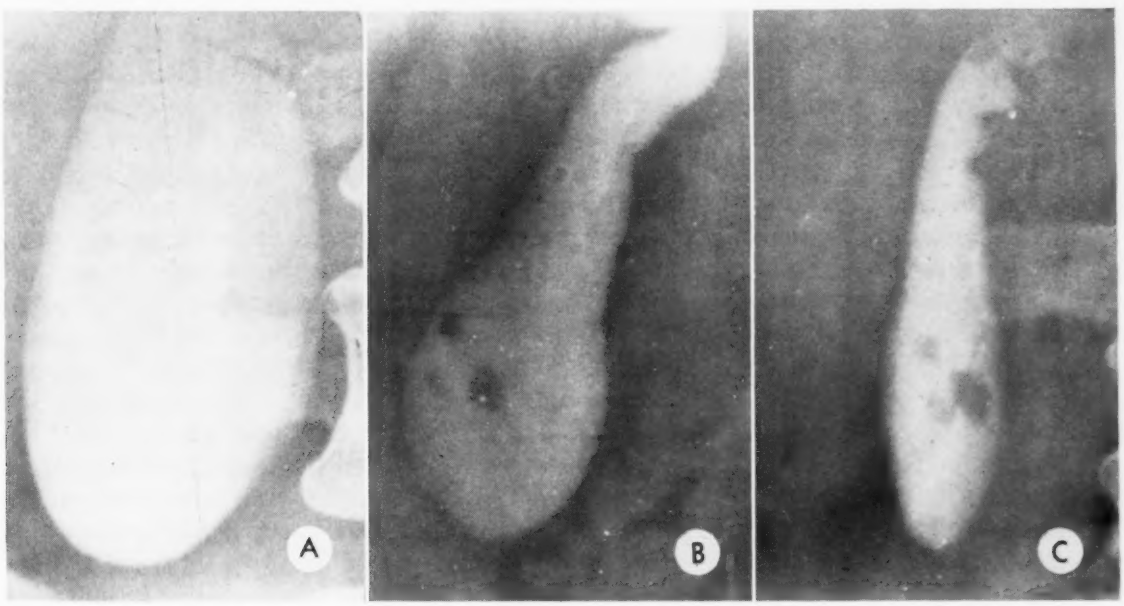

Fig. 6. (*A–C*) Disseminated multipolypoid cholesterolosis. The polyps became visible during evacuation of the gallbladder.

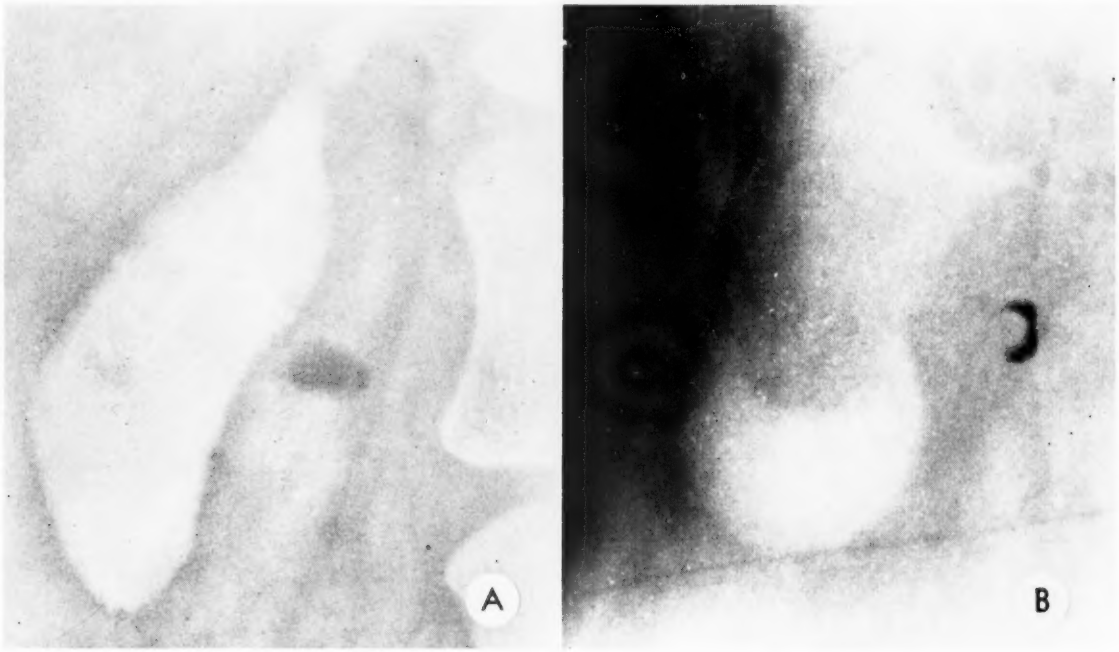

FIG. 7. (*A*) Solitary polyp proved to be a true papilloma (benign neoplasm). (*B*) Large solitary filling defect, corresponding to an adenocarcinoma.

During the last quarter century radiologists have made the diagnosis of papilloma whenever they chanced to observe tiny and fixed filling defects. Samuel and Komins[29] (1957) pointed out that the presence of several small translucent dots not due to calculi is indicative of cholesterol excrescences rather than true papillomas. Personally, we recall only one case where the gallbladder contained several well individualized papillomas.

Cholecystography reveals the presence of cholesterolosis and concomitant hyperplasias by direct and indirect signs: the first pertain to the morphology of cholesterolosis, the second to the functional disturbances. The indirect signs of cholesterolosis are usually produced when this condition is combined with other types of cholecystoses. The direct signs vary, of course, with the extent of the process or, if one prefers, with the size and shape of the individual cholesterol mass.

Pedunculated and sessile polyps are considered in a single class; micronodules and microstriations in another.

Pedunculated or sessile polyps are visualized in the iodized bile as either a notch on the contour of the silhouette or a translucent spot within the opaque area (Fig. 2, *A* and *B*). The notch corresponds to the attachment of the polyp on the wall; it is seen only in proper tangential views. A sessile polyp with a large base does not create the same difficulty in diagnosis as the one with a long and thin stem. Hefke wrote that, in a tangential projection, a stone is separated from the mucosa by an opaque isthmus. The statement is debatable since the filiform pedicle of some polyps may be several millimeters in length and may not show through the opaque line of separation. On the other hand, certain stones are partially embedded in the mucosa and then an opaque interval between the calculus translucency and the peripheral outline is not demonstrated.

The density of filling defects varies with the volume of bile displaced by the polyp and also with the concentration and amount of surrounding iodized bile. Kirklin[18] stated that the transparent area

created by what he called a papilloma is lighter than that produced by a stone. The assumption is that this is due to the greater quantity of mineral salts contained in stones; noticeable density differences are not likely to be produced by such small bodies. Transparent areas may also be caused by overlapping gas bubbles in the digestive tract, especially in the duodenum, and may lead to misinterpretation. Doubts can be removed with proper position and rotation of the patient and introduction of barium in the disturbing segments of the digestive tract (Fig. 1).

The image of the polyp may be obscured in a gallbladder distended with opaque bile (Fig. 6*A*) but it reappears during evacuation (Fig. 6, *B* and *C*) or under the thinning effect of a compression object. High voltage techniques and slight overexposure may be necessary to bring out the minute translucent shadow through the overconcentrated medium.

The size of a polyp is evaluated in relation to the gallbladder surface. Roentgenographic enlargements are of little help; when the size of a nonlithiasic translucent shadow exceeds 1 cm. (Fig. 7*B*), it is likely to be caused by a malignant growth because cholesterol polyps, papillomas, adenopapillomas and granulomas usually do not reach such a dimension.

The shape is likewise not as perfectly round as Kirklin claimed. The semitransparent shadow produced by any benign growth presents irregular contours and sinuous streaks, indicative of pits and ridges over its surface (Fig. 7*A*). A filling defect that is apparently rounded may become lobulated with compression or when the viscus is partly evacuated.

Cholesterol polyps are found in all parts of the gallbladder and even in the fundus, in which Kirklin felt that only adenomas were to be found. He regarded the midportion of the viscus as the elective site for papillomas. In our experience, true papillomas and granulomas as well as solitary cholesterol polyps were mostly seen in the middle third of the gallbladder.

A fixed position constitutes the best differential sign between a polyp and a stone or a gas bubble. However, a pedunculated polyp may bring about the illusion of displacement to a certain extent.

The number of lesions is of great significance in the differential diagnosis. Papillomas, papillary adenomas and granulomas are usually single lesions, whereas cholesterol polyps are multiple (Fig. 5, *A* and *B;* and 6, *B* and *C*). Since this multiplicity is a strong index in favor of cholesterolosis, it is important to take roentgenograms during the various stages of emptying and with varying technical factors. On a purely statistical basis, gallbladder polyposis could be diagnosed as a manifestation of cholesterolosis and this would prove to be correct in nearly one hundred per cent of the cases.

The multipolypoid type of cholesterolosis is also characterized by the fact that the polyps exhibit manifold sizes and their distribution over the opacified surface follows no definite pattern (Fig. 5, *A* and *B;* and 6, *B* and *C*). The barely perceptible lesions measure a fraction of a millimeter; the larger ones may be close to a centimeter in diameter but rarely more. Contraction of the gallbladder wall can give rise to a localized large filling defect containing several vegetations which prove to be of small size when the specimen is opened.

In summary, we wish to stress that multiplicity of small filling defects, irregularity in size and unevenness in distribution throughout the gallbladder area constitute the fundamental triad by which the roentgenologic diagnosis of cholesterolosis can be made at present.

A pertinent question is whether the nonpolypoid micronodular and disseminated form of cholesterolosis can also be detected roentgenologically. In favorable circumstances, it is possible to distinguish the minute irregularities which the cholesterol pin-point elevations impart to the mucosal pattern (Fig. 4, *A* and *B*). Whenever granulations, striations or thickened folds can be seen, they produce very fine indenta-

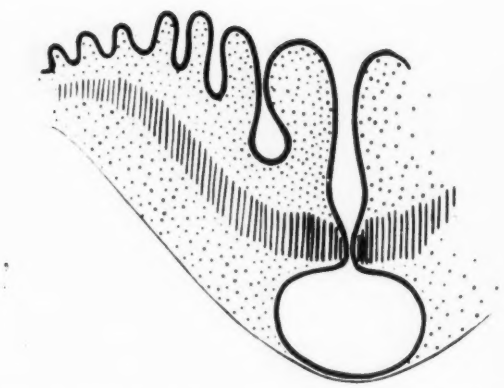

Fig. 8. Sketch of the outstanding features of adenomyomatosis. Formation of hypertrophied villi, ducts, sinuses and diverticula.

tions in the contour of the gallbladder shadow and radiolucent specks on the opaque background. The stippling corresponds to the seeds of the strawberry gallbladder. These discrete findings gain more diagnostic significance when they are accompanied by the hyperfunctional complex.

ADENOMYOMATOSIS

The three outstanding features of this type of hyperplasia (Fig. 8) are proliferation of epithelium, increased thickness of the muscle layer and formation of outpouchings of the mucosa into or through the muscularis (Luschka, Rokitansky, Aschoff).

These epithelial downgrowths may be tubular (ducts), branched (crypts, sinuses) or saccular (diverticula). The overdevelopment of the epithelium gives the mucosal membrane a glandular appearance and the deeply seated diverticula may resemble microcysts (Fig. 9). Such similarities prompted the designations of cholecystitis glandularis proliferans (King and Mac Callum[17]) and cystic cholecystitis (Bodnar[5]). This glandular and cystic resemblance is not to be confused with true glandular heterotopias, heteromorphoses (Nicholson), metaplasias of an intestinal type (Brunner or Lieberkühn glands) or even of gastric or pancreatic type, which can all form localized excrescences called choristomas. True mucous glands, existing normally only in the infundibulum, heterotopic tissue and the surface epithelium, by metaplasia may proliferate and give rise to true benign mucosal neoplasms, such as adenoma and adenopapilloma, which carry a high potentiality of malignant transformation.

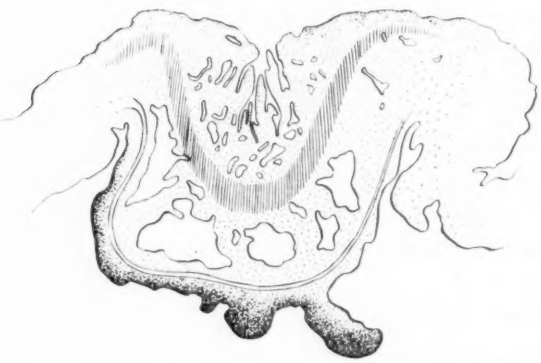

Fig. 9. Sketch of adenomyoma. The mucosal membrane has a glandular appearance and the deeply seated diverticula resemble microcysts.

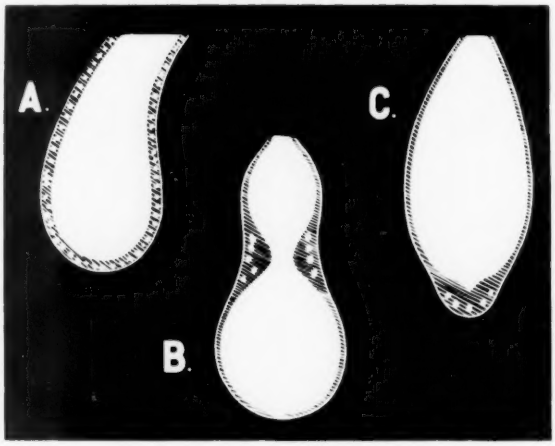

Fig. 10. Diagrams of types of adenomyomatosis. (*A*) Generalized type. (*B*) Segmental type. (*C*) Localized type (adenomyoma).

GENERALIZED AND SEGMENTAL TYPE DIFFUSE ADENOMYOMATOSIS

The diffuse form of adenomyomatosis may involve the entire gallbladder (Fig. 10*A*; 11, *A–E*; and 12), annular segments (Fig. 10*B*; and 13) or ill-defined parietal zones. Whatever the type of distribution of adenomyomatosis, the cholecystographic diagnosis was based hitherto only on the visualization of intramural diverticula.

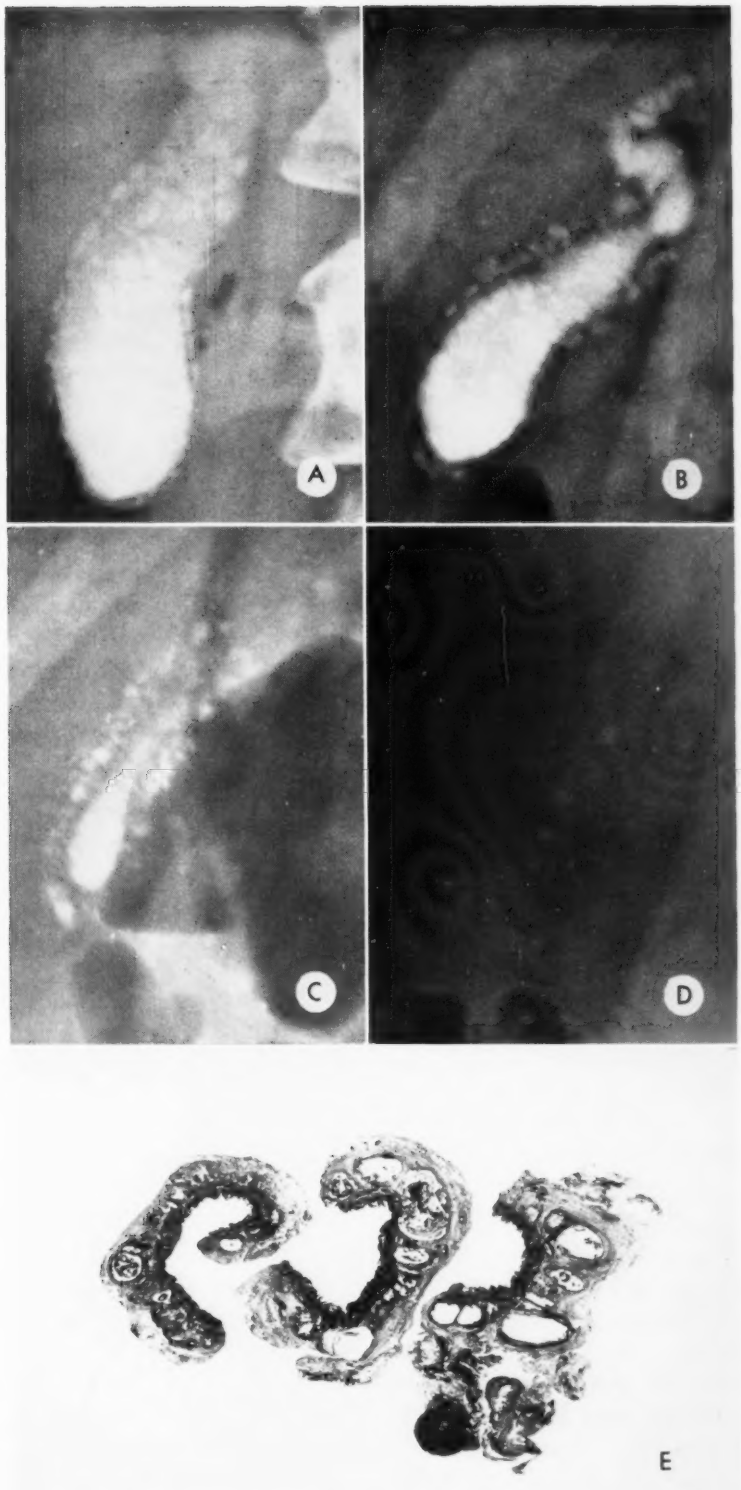

FIG. 11. Adenomyomatosis; generalized type. (*A*) A fully distended and normally shaped gallbladder presenting numerous diverticula over the surface and around the edge. (*B*) Same gallbladder exhibiting a contraction ring that gives a piriform shape to the shadow. The annular narrowing obviously is not an organic abnormality. (*C*) The diverticula are blown up by intraluminal pressure. The interspace separating them from the main cavity indicates the thickness of the muscular coat through which communicating ducts are seen. (*D*) A preliminary roentgenogram before the partaking of the opaque medium reveals discrete opaque spots corresponding to concretions in the diverticula. (*E*) Sections of the operative specimen show numerous diverticula and intramural concretions.

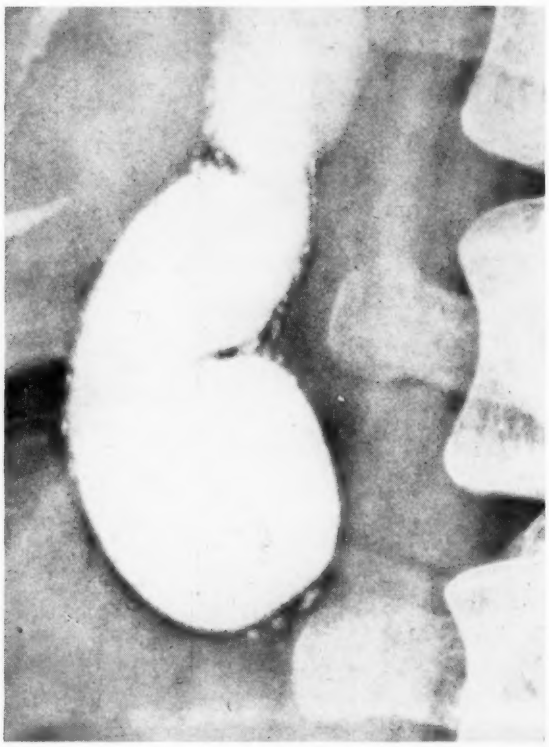

FIG. 12. Generalized adenomyomatosis with two thickened folds producing a triloculated gallbladder. (Courtesy of Dr. Joseph O. Reed, Detroit.)

These diverticula, when filled with iodized bile, appear as a chain of opaque dots at a short distance from the periphery of the gallbladder shadow; they remind one of a map showing minute atolls along a coast (Fig. 11, *B* and *C*). Here and there, opaque filaments reveal the communicating ducts between the small marginal sacs and the gallbladder lumen. The interspace separating these spots and the main cavity represents the thickness of the intervening muscle layer in different states of rest or contraction (Fig. 11*C*). Naturally, the interspace decreases in width as the gallbladder wall stretches and, conversely, it increases during emptying contraction.

The shadow of the gallbladder is not uniform and opacified diverticula can show through as additional specks. Tiny marginal indentations and transverse striations may also be observed; they represent excessive folding of the thickened mucosa with added muscular wrinkling. Calcified concretions are at times found within the diverticula (Fig. 11*E*). The intramural concretions are rarely diagnosed as such on cholecystograms; they should be sought for before the contrast medium is administered (Fig. 11*D*). This procedure, however, is not always followed since it is still believed that the concretions cannot be visualized, which is not always the case.

The roentgenologic literature concerning the visualization of intramural diverticulosis is restricted to sinuses and pouches containing radiopaque material. Occasionally, gas in pneumogenic cholecystitis may demonstrate the Rokitansky-Aschoff sinuses. Whenever parietal pockets are conveniently distended by gas due to anaerobic bacilli, they appear as transparent blebs around the gallbladder cavity which is also pneumatized (Åkerlund and Rudhe,[2] Tedesco, Jutras and Gernez[13]). The size of

FIG. 13. Adenomyomatosis; segmental type. The locally thickened wall produces a narrowing.

these gaseous blebs is much larger than that of the liquid-filled diverticula due to the rupture of an overdistended membrane and diffusion of gas in interstitial tissues (Fig. 14).

In 1957, Culver, Berens and Bean[8] studied the 20 cases of intramural diverticulosis recorded in the literature with 5 additional cases of their own. Since 15 out of 20 gallbladders about which complete information was available showed a "definite zone of stenosis or constriction in the proximal portion," the authors concluded that a circular stricture is "the most important etiological factor."

Our own observations do not support the above conclusion which we feel was based on insufficient roentgenologic data. Roentgenograms taken over the entire period of emptying clearly demonstrate that a piriform narrowing in the upper half of the gallbladder disappears during periods of muscular relaxation (Fig. 11, *B* and *C*). The transitory character tends to prove that large rings (Fig. 12) are the result rather than the cause of adenomyomatosis; they are due to irritability and overdevelopment of the muscular layer. Permanent and semivalvular kinks at the proximal end of the gallbladder as well as at the phrygian cap mucosal folding at the distal end are a different matter. These represent organic malformations and certainly are contributing factors.

Contrary to what is often stated, diverticula may be found proximal or distal to a stricture (Fig. 11*B;* and 12). The mere fact that diverticula exist at the same level or proximal to stenotic rings proves that their pathogenesis is more complex and one is reminded of the colon where a localized spasm with secondary fibrous hyperplasia in a given segment does not explain the formation of diverticula distally.

In general, it is only on cholecystograms taken in the later phase of evacuation that diverticula come to light, more numerous and larger. Here, as in the large bowel, they attain their maximal dimensions in areas of contraction. At times, the opaque filled

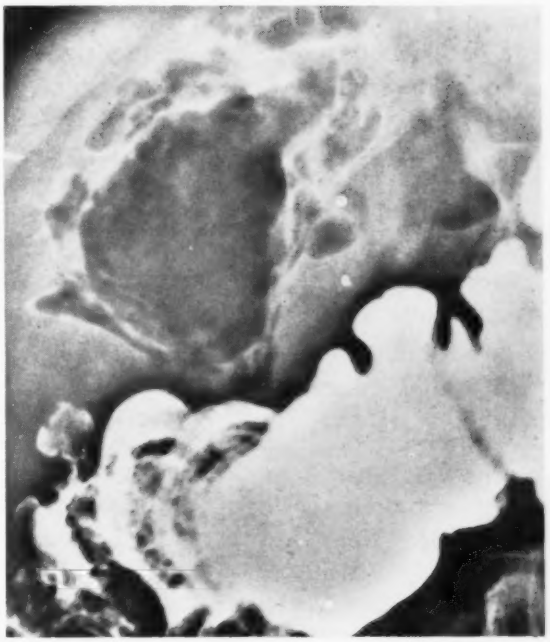

Fig. 14. Gas blebs around the gallbladder cavity that itself is inflated by acute pneumogenic cholecystitis. Diverticula are also seen on the large bowel, a frequent coincidence.

diverticula at the periphery resemble the pattern of a string of pearls.

The study of the mucosal outline is also helpful even when the presence of diverticula dispels all doubt concerning the existence of adenomyomatosis.

Cholecystograms correctly taken at a favorable time of evacuation, under controlled pressure, and especially if printed later on the LogEtron can reveal serrations strikingly similar to the "spiked" or "saw-toothed" appearance of spasmodic diverticulosis of the colon. The mucosal folds run transversally in the body of the gallbladder but appear fan-shaped when they occur in the fundus. To grasp the substratum of these rugae or striations, it must be remembered that the gallbladder possesses no muscularis mucosae or submucosa. The lamina propria of the mucosa is thus directly anchored to the muscular layer. The shape, size and direction of the folds and villi thus depend on the state of contraction and also on the presence of hyperplasia.

The indentations of the gallbladder wall,

which form shallow or deep kinkings, as in the phyrygian cap, were so frequently seen in our material that we now hesitate to consider these as characteristic of malformations. These kinkings do not exclude adenomyomatosis; in fact, they are one of the preferred sites of the process. The folds increase in size under the dual influence of hyperplasia and muscle contraction. Normally these congenital kinkings are thin and smooth; their thickness does not exceed 2 mm. as they simply consist of two epithelial layers held back to back by the subepithelial connective tissue. Any increase in width is an indication of some pathologic process. The semilunar kinkings near the fundus that constitute the phrygian cap should not be our sole consideration. The pseudovalvular folds of the infundibulocystic region, although more common and of smaller size, are significant if minimal signs of gallbladder hyperplasia are to be discerned.

The segmental type of adenomyomatosis (Fig. 10 *B;* and 13) is unpredictable as to location. It may occupy the infundibulocystic area, a large portion of the fundus, the body or several segments separated by relatively unaltered regions.

In the gallbladder as in the colon, mucosal herniations are evidence of some weakness in the muscular layer. The outpouchings of the epithelium, that develop because of increased intraluminal pressure, can only occur between the interstices of muscle bundles. Beyond the muscular layer these outpouchings bulge and form rounded diverticula. When these diverticula are large enough and sufficiently opacified, the radiolucent line that separates them from the gallbladder lumen is an index of the thickness of the muscular layer. Serial roentgenograms demonstrate that this layer remains elastic, its proportions changing with the quantity of bile in the viscus. The narrow ducts that connect the diverticula to the lumen are often visible as thread-like opaque lines.

Adenomyomatosis of the gallbladder and diverticulosis of the colon are analogous not only in the distribution and morphology of the pockets but also in the intervening strictures. To this may be added the fact that both conditions often coexist in the same individual, a phenomenon that increases with age.

Congenital foldings of the wall, acquired kinkings or narrowings, partial obstructions by stones, reflex spasms, or, in short, any permanent or temporary obstacle to the emptying of bile promotes the development of these lesions, accelerates and intensifies their formation and guides their progress.

Angulations, strictures, and pseudovalves had been mentioned by Berg (1920) and Rossiter (1938) as factors responsible for gallbladder dyskinesia. However, the exact role played by these morphologic peculiarities of gallbladder physiopathology was only recognized when radiomanometry during or following surgery became available.

LOCALIZED TYPE ADENOMYOMA

The term "adenomyoma" used for the first time by Sutherland[30] in 1898 to designate a small and hemispheric nodule bulging in the fundus of certain gallbladders is objectionable for two main reasons: first, the growth is not a neoplasm; secondly, most of the glandular-like elements within the small mass do not constitute and do not derive from true glands.

Nevertheless, the name fits in our nomenclature of cholecystoses (Table 1 and Fig. 10*C*) since it indicates a circumscribed and tumor-like form of adenomyomatosis.

Roentgenologic semeiology of adenomyoma has not progressed to any great extent since Kirklin[18] briefly outlined the crescentic filling defect in the fundus of the gallbladder (1931 and 1933). Radiologists are aware of intramural diverticula only on the basis of about 40 cases published since March's[23] article in 1948. On the other hand, roentgenologic data on diverticula within adenomyoma were not available before our own early studies in

TABLE III

ADENOMYOMATOSIS

JANUARY, 1957–JULY, 1958

Cholecystography	6,082
Cholecystectomy	982
No. of Cases with Adenomyomatosis (pathology report) (all with stones except 72 cases)	326
No. of Cases with Adenomyomatosis plus Cholesterolosis (pathology report)	32

Note: The above figures are representative of a period during which radiologists, physicians, surgeons and pathologists as a group showed special interest in the subject. When stones are demonstrated by cholecystography, whatever the radiologist may say about the gallbladder wall is considered purely academic. During the stated period 21 gallbladders removed because of adenomyomatosis without stones confirmed the preoperative roentgenologic diagnosis.

1956.[14] Since pathologists never insisted on the fact that adenomyoma is simply a localized form of adenomyomatosis, radiologists were not properly guided in exploring the problem with their own means.

Sutherland[30] in his brief article of 1898, which included a single autopsy specimen, described in a few lines the intraluminal appearance of adenomyoma, but the most thorough anatomic study of this lesion was made by Nicod[27] in two articles (1922 and 1927), unsurpassed in subsequent publications.

In 1957, LeQuesne and Ranger[20] reported 12 cases of cholecystitis glandularis proliferans, 4 of which were discovered incidentally at autopsy. In some of these gallbladders, there was thickening of the wall with diverticula at the fundus. This finding influenced the authors to state that hyperplasia in the fundus was of the same nature as the diffuse or generalized form of cholecystitis glandularis proliferans. Their statement thus brought support to the theory we had already proposed at American and European meetings.

Since we have learned to recognize the elusive features of adenomyoma of the fundus on cholecystograms, we have succeeded in detecting this condition in no less than 5 per cent of the gallbladders examined in our department (Table III). Such a high rate of positive roentgenologic diagnosis casts some doubt as to whether the lesion has any clinical significance and whether surgery is indicated in these cases.

Histologic description. As is known, formalin fixative produces on muscles and pliable tissues of anatomic specimens effects that are quite comparable to contraction of the living organ. A photomicrograph of an axial section of an adenomyoma containing gallbladder, retracted by the formalin fixative, readily demonstrates a clubbing of the distal end of the viscus and also shows that all the constituent tissues contribute to the enlargement (Fig. 15, *A–C*). The localized mural thickening manifested by the adenomyoma is not limited by a connective tissue capsule. Although the lesion is not sharply demarcated from the surrounding normal tissue by a distinct membrane, it is rather well defined by a sudden enlargement of all layers of the gallbladder wall beginning a few millimeters above the tip of the fundus. All histologic coats participate in this process: mucosa, muscular layer, adventitia and even the serosa. The hyperplastic mucosa confined to a restricted space at the tip of the contracted fundus curls up into a rounded mass that has a glandular appearance (Fig. 15*C*). In fact, the excess mucosa that is forced to occupy a limited area develops folds that resemble tubular or branching glands. The muscular layer is traced at a gradually increasing distance from the intraluminal outline. In this fashion the muscular membrane describes an inverted arc, the diameter and depth of which vary in accordance with the size of the mucosal bud. The inner outline of the muscular arc is relatively sharp and regular in opposition to the outer aspect which is broken up into fascicles of variable sizes that fan out like tentacles around the diverticula before reaching the fibrotic spurs arising from the adventitia. The plexiform arrangement of the muscular bundles facilitates the passage of epithelial expansions. These herniations form cavities on the outer surface of the

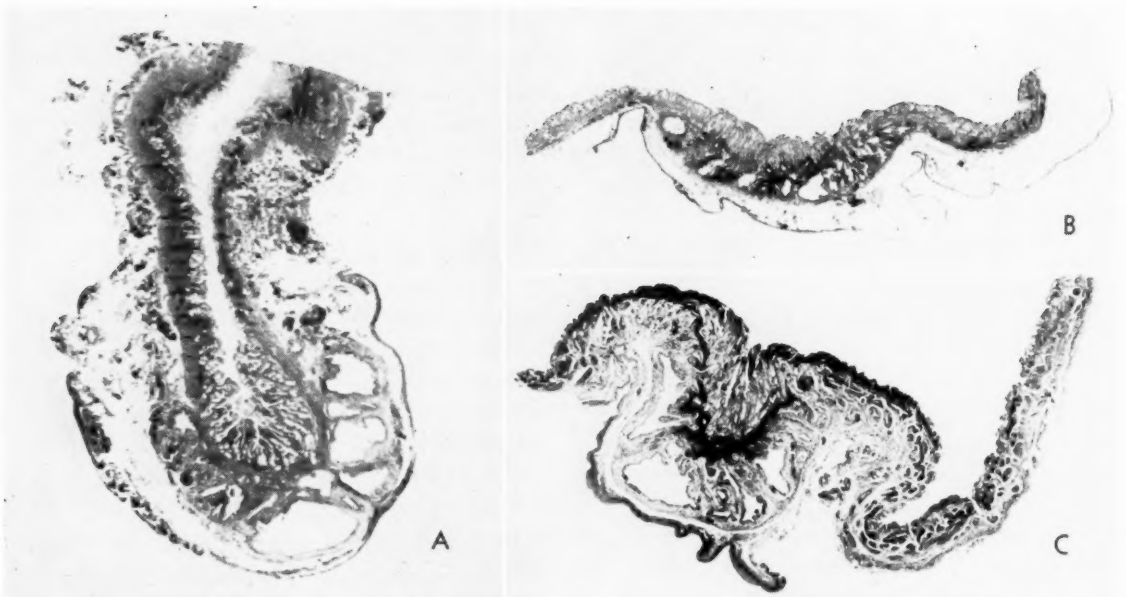

FIG. 15. (*A*) Axial section of the gallbladder through a focus of adenomyoma localized in the fundus. The clubbing of the hyperplastic mucosa at the lower end of the sac leaves the impression that the muscular coat underneath it is extruded. The muscular layer describes a concave arc and below it large diverticula are seen also along a curved line. (*B*) There is a folding of the entire wall at the edge of thickening, indicating that invagination is in the making. (*C*) The invagination is completed and the node of adenomyoma is formed.

muscular coat. Rounded or loculated, they may measure several millimeters in diameter and become visible to the naked eye. At times, they strongly resemble small cysts, an impression that explains the term "cholecystitis cystica" used by Bodnar.[5] The outer connective tissue layer (adventitia) and the serosal peritoneum that covers entirely or in part the fundal hyperplasia can also undergo fibro-adipose thickening.

Although the above microscopic observations are incomplete, they nevertheless reveal that the pseudotumoral hyperplasia of the gallbladder fundus that we call adenomyoma is absolutely identical with the tissue changes characteristic of adenomyomatosis (cholecystitis glandularis proliferans).

The preceding description applies to an adenomyoma that is extruded from an empty and contracted gallbladder. However, contrary to this evagination, the mass can also lead to invagination into the cavity of the viscus. Depending on the degree of invagination, the adenomyoma will vary in height and consistency.

At the maximum of extraluminal protrusion, microscopic sections show that the muscular layer forms an arc, the concavity of which is filled with an excess amount of mucosa. The latter follows the concave line of the muscular layer and thus forms a dimple toward which the epithelial ducts converge.

The adventitia and serosa also participate in the production of perinodular folding. In this case, a more or less deep and circular furrow delineates the base of the mass on the external side of the gallbladder.

Roentgenologic Manifestations. The adenomyoma of the fundus of the gallbladder presents on roentgenologic examination a manifold appearance, depending on the degree of filling of the gallbladder (Fig. 16, *A-D*), the changing anatomo-physiologic status (Fig. 17, *A-D;* and 18, *A* and *B*) and the position or projection in which the examination is made. The most valuable information is derived from examinations

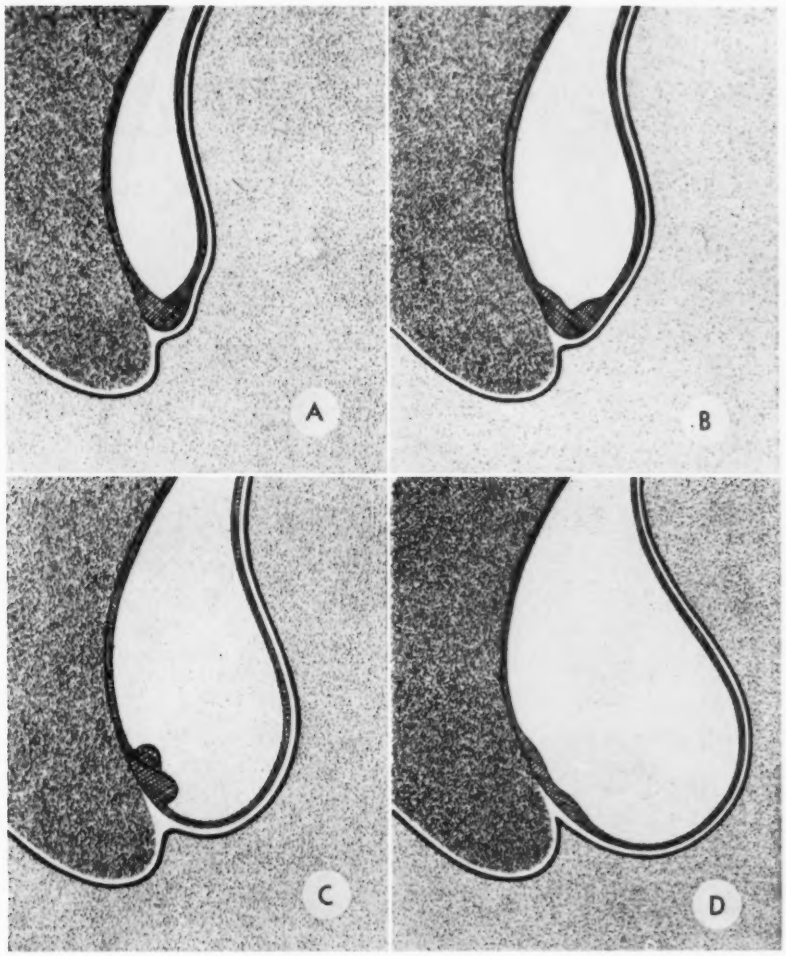

Fig. 16. Diagrams illustrating changes in shape, size and location of an adenomyoma relative to the degree of filling of the gallbladder. (*A*) With a slight degree of distention of the biliary sac, the adenomyoma has sufficient room in the hepatoperitoneal space and remains in the evaginated state. (*B*) As filling increases, the reduction of space tends to push inward the node of hyperplasia. (*C*) The adherent portion of the gallbladder holds the adenomyoma at the edge of the hepatic fossa while the peritoneal hammock expands; in this manner, the small mass is pressed against a hard surface and invagination is produced. (*D*) Excessive filling stretches eccentrically the entire gallbladder wall including the adenomyoma focus. The latter thus becomes wider and less protuberant.

made in (1) the frontal and (2) the profile views (Fig. 18, *A* and *B*).

(1) In the frontal view, one may distinguish an: (a) intraluminal, (b) transmarginal and (c) extraluminal appearance of the adenomyoma (Fig. 18*A*).

(2) In the profile view, one can demonstrate the anatomo-physiologic status of the mass, *i.e.*, (a) whether it presents invagination, (b) is in an intermediary state or (c) has undergone evagination (Fig. 18*B*).

(1.a) In the frontal view, a totally circumscribed intraluminal adenomyoma appears as a rounded filling defect projecting through the opaque material. In the center of the filling defect, there usually is a dot that varies in size and density, depending on the dimensions of the apical crater it represents. The image strongly resembles a life buoy or a cholesterol calculus with a mineral nucleus (Fig. 19*A;* and 20 *A–C*). We know of at least 2 cases in which an image of this kind has been interpreted as a

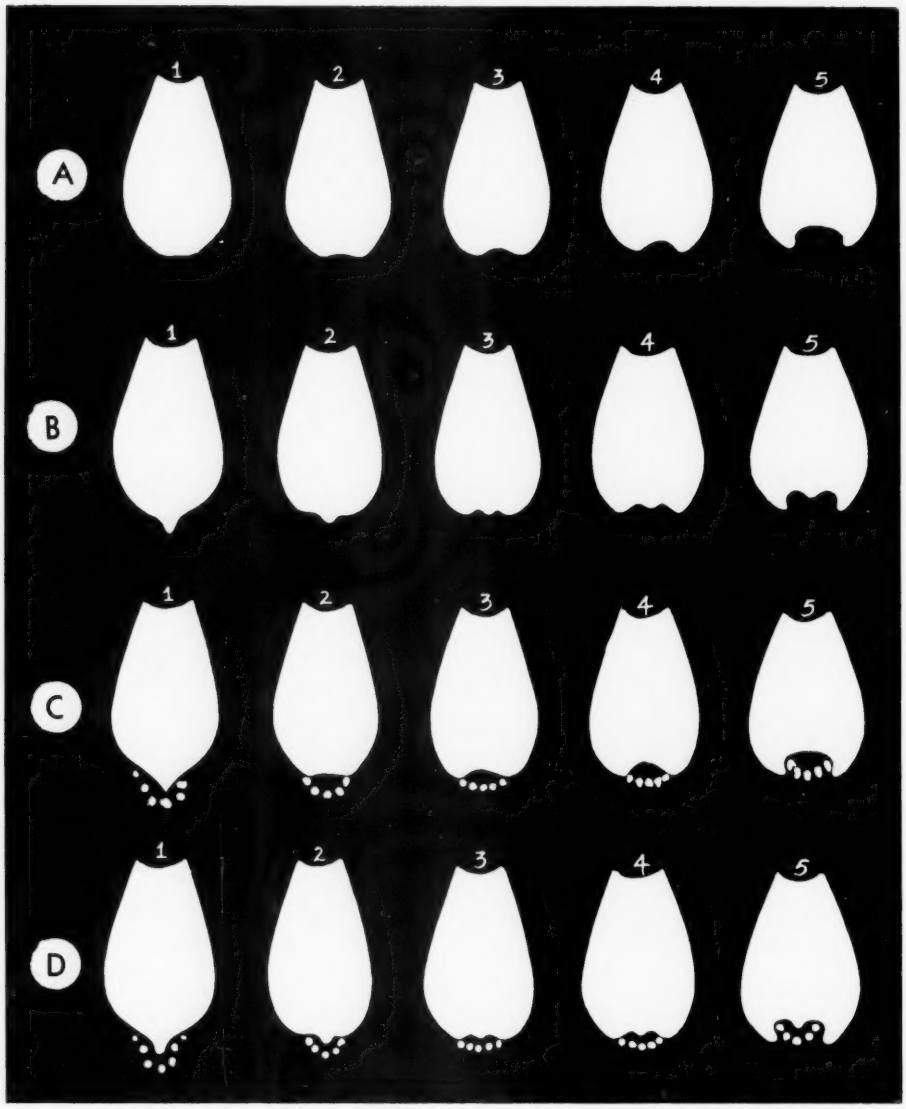

FIG. 17. Diagrammatic illustration of the various roentgen images produced by adenomyoma in the tangential views. (*A*) The slight notching at the tip of the fundus of the gallbladder increases from 1 to 5 to become a crescent shaped defect. (*B*) A gradually enlarging central crater develops, culminating in the omega sign. (*C* and *D*) Filled diverticula add a string of opaque specks on the external side of the notch in *A* and crater in *B*.

gallstone. Whenever there is adequate filling, the diverticula visualize as specks inside the translucent ring, giving the appearance of bullet marks around the eye of a target. Although differential diagnosis from a calculus can be made by the movability of the latter, one must be cautious since, as already mentioned, adenomyoma also has a certain mobility.

(1.b) In the transmarginal location (Fig. 19*B* and 21, A–*C*), the adenomyoma may be represented simply by a semilunar concavity of the crater and the diverticula may not contain any opaque material. This crescentic filling defect has been described by Kirklin.[18] A funnel-shaped spot may be added to the center of the above arc and cause a design that may be compared to what is called an "organ point" in musical notes (Fig. 21*C*). The tip of this funnel-

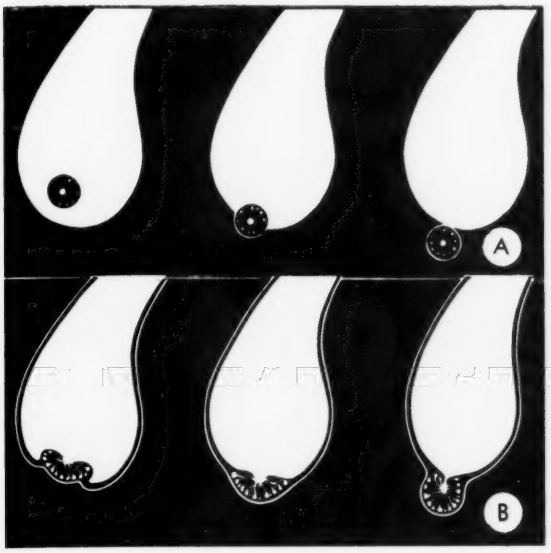

Fig. 18. Diagrams of the roentgen images of adenomyoma in the frontal and profile views. (*A*) Frontal view. *Left:* intraluminal; *Middle:* transmarginal; *Right:* extraluminal. (*B*) Profile view. *Left:* invagination; *Middle:* intermediary state; *Right:* evagination.

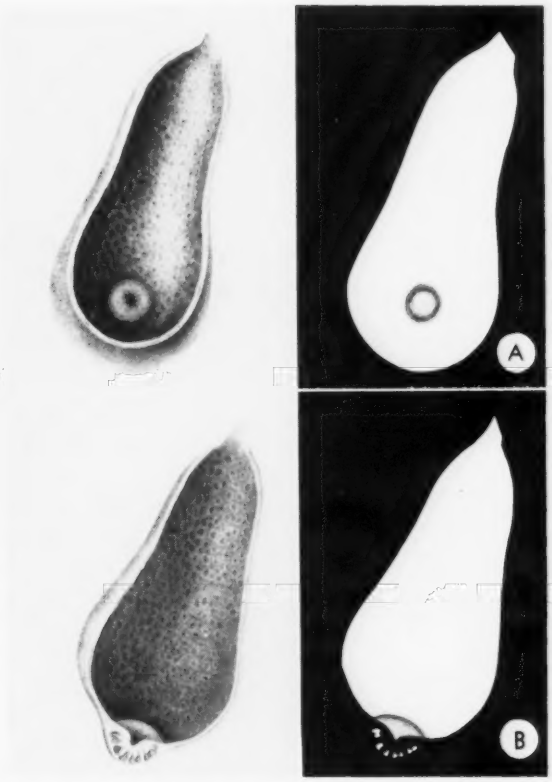

Fig. 19. Diagrams of adenomyoma. (*A*) Intraluminal type giving pseudo-calculus appearance. (*B*) Transmarginal type showing all elements: crater, collar, diverticula.

shaped dot is directed outward. The crescentic filling defect may contain a series of small opaque flecks distributed on a curved line that bridges both sides of the gap of the gallbladder contour. The flecks represent diverticula in the peripheral layer of the adenomyoma.

(1.c) In the extraluminal adenomyoma, the typical appearance is seen when the body of the opacified gallbladder is separated from the fundic adenomyoma either by a congenital fold, referred to as a phrygian cap, or by a localized thickening

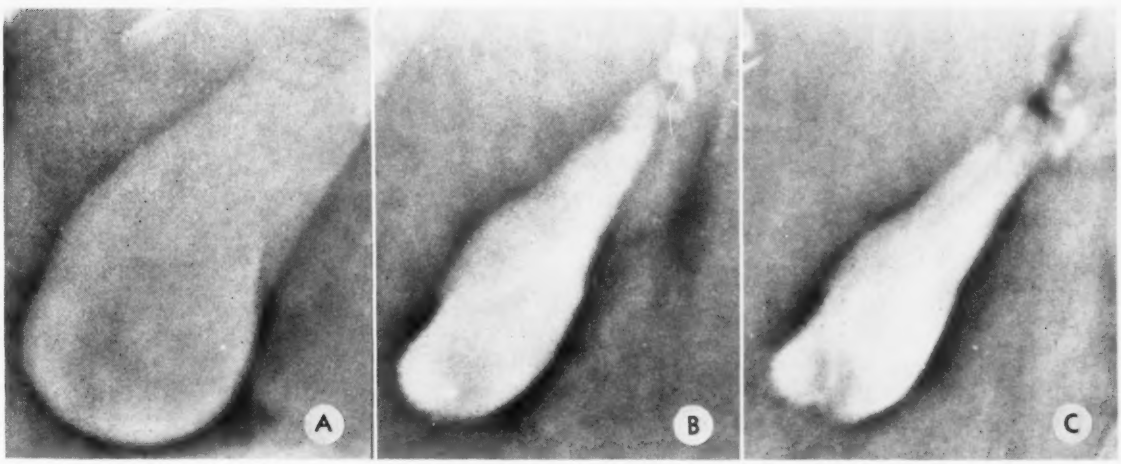

Fig. 20. (*A*) Intraluminal adenomyoma showing pseudo-calculus appearance. (*B* and *C*) After partial evacuation the appearance changed into one of transmarginal type.

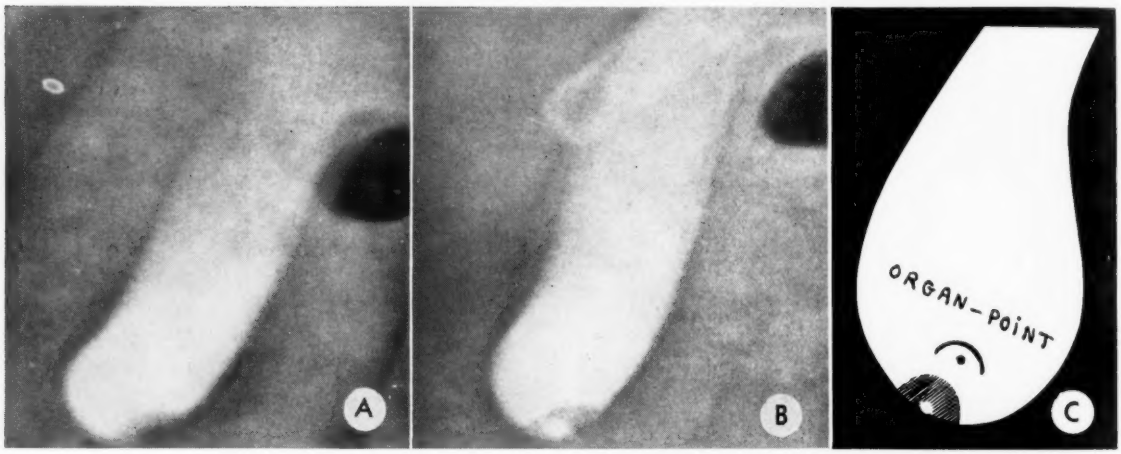

FIG. 21. Transmarginal adenomyoma. (*A*) Unfilled crater. (*B*) Crater and diverticula opacified. (*C*) Diagram illustrating resemblance to the "organ point" in musical notes.

of the gallbladder wall in the lower portion where some opaque material might be present or completely absent. In such an instance, the central crater is filled with opaque material and is surrounded by a circular series of little dots corresponding to the communicating diverticula. This special arrangement recalls an architectural rosette or an ancient monument in Brittany called "Cromlech" (Fig. 22, *A–D*).

(2.a) In the profile view, when invagination is present (Fig. 23, *A–F;* and 24, *A–C*), the circular groove outlining the base of the mass forms an angle that gives a horny appearance to the marginal notch. This is quite similar to the picture commonly seen in lateral roentgenograms of a pedunculated tumor submerged in barium in the digestive tract. The top line of the indentation may be slightly flattened and more or less depressed in the middle. It is customary for us to refer to the design as the everted omega sign. When some of the extramuscular diverticula are filled with iodized bile, they form a string-like pattern of opaque pearls ornamenting the neck of the marginal notch.

(2.b) The intermediary state of adenomyoma may comprise aspects ranging from full protrusion into the lumen of the gallbladder to marked extrusion. Accordingly, the roentgenologic pattern may vary considerably, as illustrated in Figure 17, *A–D*.

(2.c) The adenomyoma mass in total evagination gives a much more discrete picture (Fig. 25, *A–D*). The funnel-shaped apical depression may change the rounded outline of the lower part of the gallbladder shadow into a slightly angular one. Whenever such an image is seen in a filled gallbladder, one must automatically search with much attention for signs of diverticula in the vicinity. The small sacs may contain only a small amount of iodine and consequently be rather difficult to perceive. We know, through long experience, that opaque bile filled diverticula below the lower contour of the gallbladder silhouette are regularly missed because of the manner in which cholecystograms are routinely studied in busy departments. An angular contour of the lower part of the gallbladder, indicates that Rokitansky-Aschoff diverticula could be present and an intent search should be made for a hemispherical extruding soft-tissue mass, the density of which is, of course, quite similar to that of neighboring organs.

The eventual discovery of an adenomyoma of the lower pole of the gallbladder must not put an end to the investigative efforts of the examiner; on the contrary, the fact that there is such a lesion should encourage the radiologist to study functional and morphologic deviations from normal as well as the possibility of involvement of

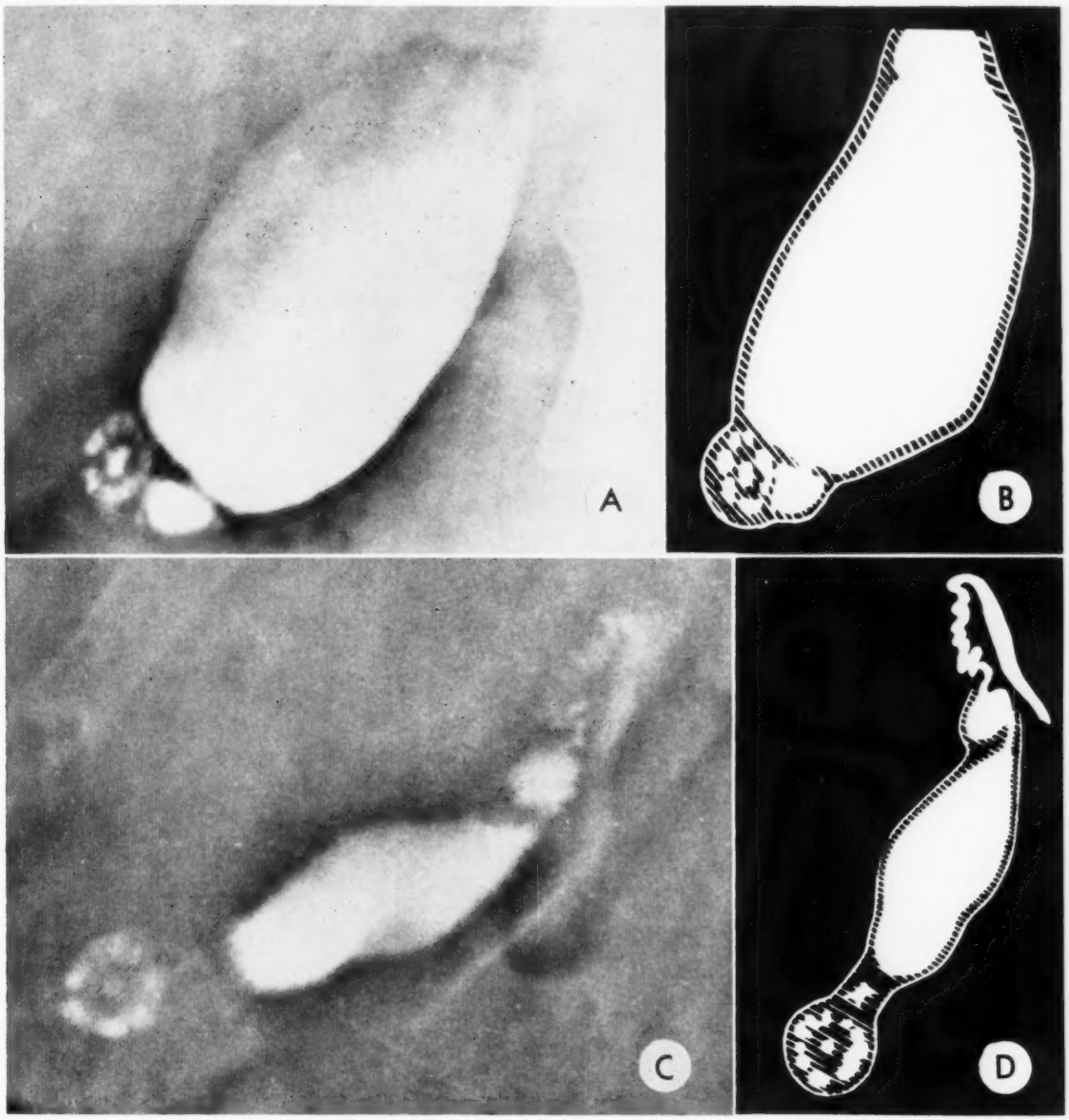

Fig. 22. Extraluminal adenomyoma. (*A* and *C*) Cholangiograms. (*B* and *D*) Respective diagrams illustrating the rosette appearance or the resemblance to an ancient monument in Brittany called "Cromlech."

other segments by adenomyomatosis, cholesterolosis or neuromatosis which, as previously emphasized, are frequently associated conditions. If roentgenologic data are not sufficient in the first series of cholecystograms to allay suspicion, the examination should be repeated and the selective technique previously described should be applied meticulously so that previously ill-defined images may become conclusive ones. At times, observations over longer periods may prove necessary (Fig. 26, *A–D*).

NEUROMATOSIS

The term "neuromatosis" designates a nonneoplastic proliferation of the autonomous nerve fibers. According to Riopelle,[28] there are two distinct types of gallbladder neuromatosis: deep and superficial. These two forms of hyperneurogenesis differ not only as to their site but

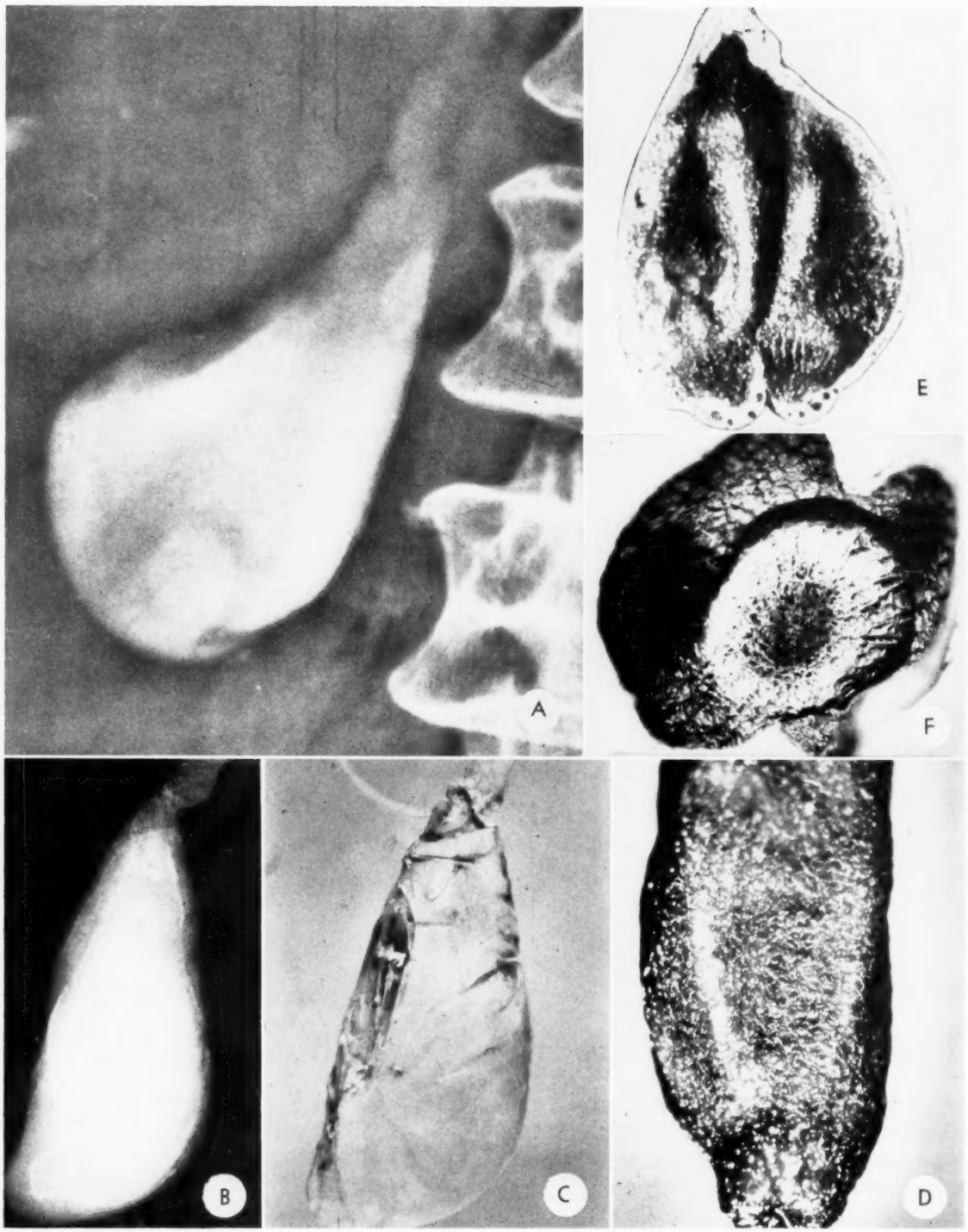

FIG. 23. (*A–F*) Complete invagination of adenomyoma (very similar to Kirklin's case[18]). Large opaque crater with few surrounding diverticula and the crescentic radiolucent rim. (*A*) Cholangiogram. (*B*) Roentgenogram and (*C*) photograph of the removed gallbladder. (*D–F*) Photographs of sections of the operative specimen.

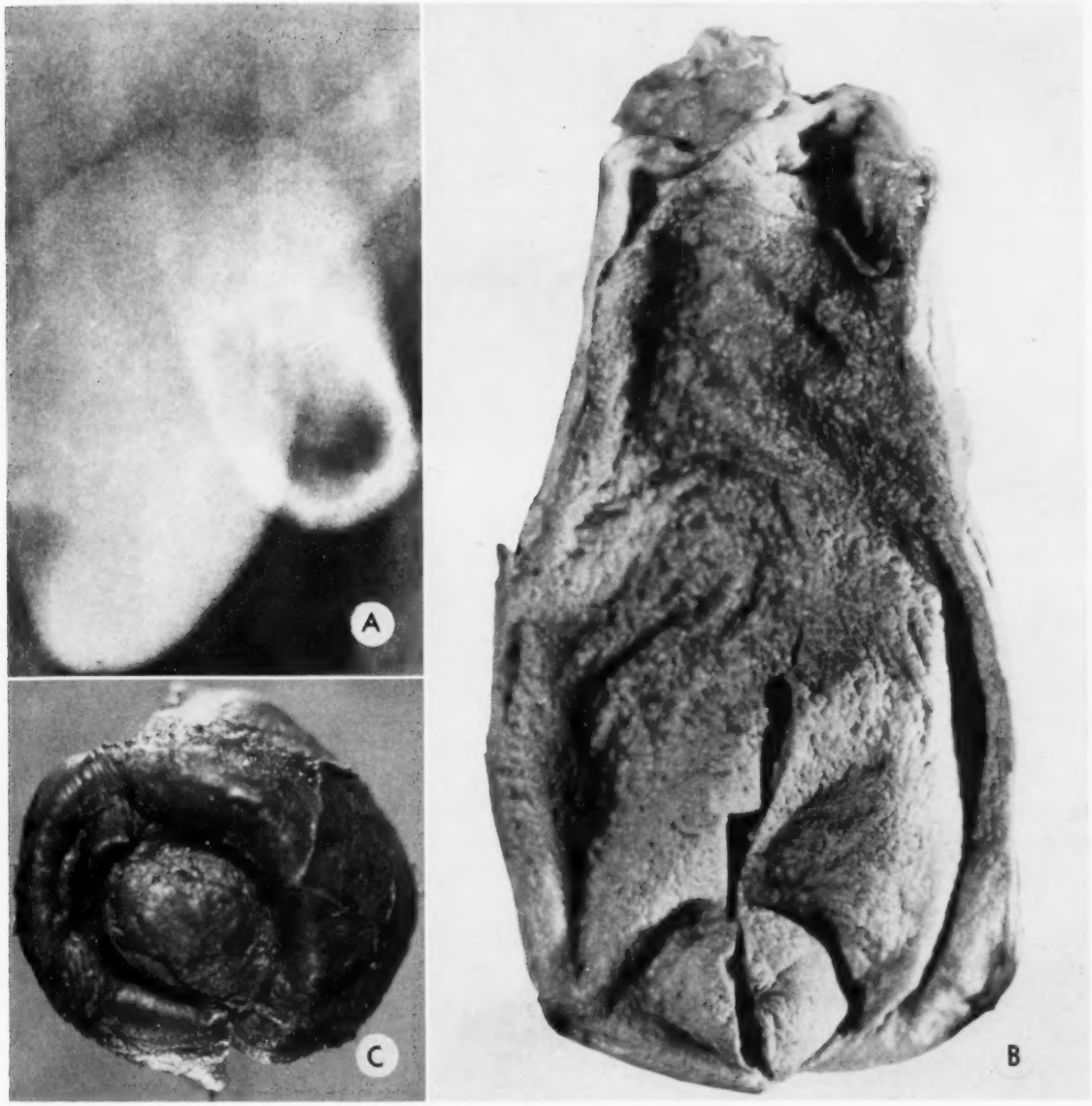

Fig. 24. (*A–C*) Partial invagination of adenomyoma. (*A*) Cholangiogram. (*B* and *C*) Photographs of sections of the operative specimen.

also in their structure and quite obviously in the provocative causes.

Deep or Adventitial Neuromatosis

This rarer type of neuromatosis has a special affinity for the perimuscular layer (adventitia) of the viscus but, at times, it also involves the muscular coat. At the writing of his original report in 1942, Riopelle[28] had found only 2 examples recorded in the literature that corresponded to his own description of "adventitial neuromatosis." These 2 cases were designated under a different name. The first one had been reported by Hamperl in 1928 and the second by Schack in 1931.

The hypergenesis of nerve fibers in the outer layers of the gallbladder is frequently associated with extensive mutilations which induced Riopelle to consider this kind of abnormal nerve proliferation not as a developmental error but as an acquired lesion due to an overregenerative reaction.

Deep or adventitial neuromatosis initi-

ated by an exuberant post-traumatic repair stimulus is mentioned here mostly to distinguish it from superficial or mucosal neuromatosis, with which it seems to have no correlation.

SUPERFICIAL OR MUCOSAL NEUROMATOSIS

Riopelle[28] was the first to describe this most interesting and still little known form of neural hyperplasia which is located in the subepithelial layer of the mucosal membrane, the lamina propria. It is commonly associated with two other forms of gallbladder hyperplasia, cholesterolosis and adenomyomatosis.

Riopelle summarizes the histologic features of superficial neuromatosis as follows: "Whereas normally the fibers of the mucosal plexus are invisible at low magnification, minimal hypergenesis is characterized by the appearance of sub-epithelial fascicles, in the upper half of the lamina propria. Higher grades of hyperplasia are denoted by cushion-like accumulations of fibers, or even nodular masses of interlacing bundles, totally replacing the connective tissue and locally swelling the folds of the mucosa in balloon-like fashion."

The microscopic recognition of superficial neuromatosis requires proper fixation of the specimen as well as special staining. Systematic studies with the aniline blue trichrome technique and, still better, with silver impregnation would prove that superficial neuromatosis is detectable in probably more than 10 per cent of surgically removed gallbladders.

Riopelle noted in his original article that he found mucosal hyperneurogenesis associated with cholesterolosis in 63 per cent of the specimens he had studied for the purpose. Due to such a frequent coexistence, the author raises the question of mutual relationship of both lesions and recommends a search for nerve hypergenesis whenever the gallbladder shows minimal gross or histologic lesions, particularly in cases with a typical history of biliary disturbances.

Riopelle considers subepithelial neuromatosis to be the result of direct irritation. He also remarks that while the superficial nervous plexus is overdeveloped and overstimulated, the deeply seated plexus remains normal as well as the connections between the two networks. Under such conditions, physiologic and anatomic factors are set up perfectly to produce reactions on the whole autonomous nervous system which can also reach levels of consciousness.

This remark helps to explain the intense pain that is recorded as the predominant symptom in many case histories of biliary disease without stones or jaundice.

From roentgenologic and anatomic findings as well, the association of neuromatosis with cholesterolosis and adenomyomatosis is estimated to be very frequent; however, nervous tissue hyperplasia is reported only when the changes are impressive enough and familiar to the examining pathologist (Fig. 27, *A–D*).

THERAPEUTIC CONSIDERATIONS

The symptomatology of cholesterolosis is a typical biliary dyspepsia with pain.

Chiray and Pavel[7] state that the "strawberry gallbladder is painful, even exquisitely painful. This pain recurs with frequent bouts of colic every two, three or four weeks." Caroli *et al.*[6] add that "these gallbladders are among the most painful." Riopelle[28] in his original work on neuromatosis stresses the frequency of the painful attacks with reflex phenomena and the severity of the pain.

From this evidence, there is ground to believe that the neural hyperplasia is the cause of the pain. In addition, adenomyomatosis, which is also a frequently coexisting lesion, causes strong muscular contractions. These contractions, added to the excessive local nervous supply, in their attempts to overcome a mechanical barrier are responsible for the colicky pain.

It thus seems logical to consider neuromatosis as a common denominator; but more detailed studies and a greater number of cases are needed. The material available

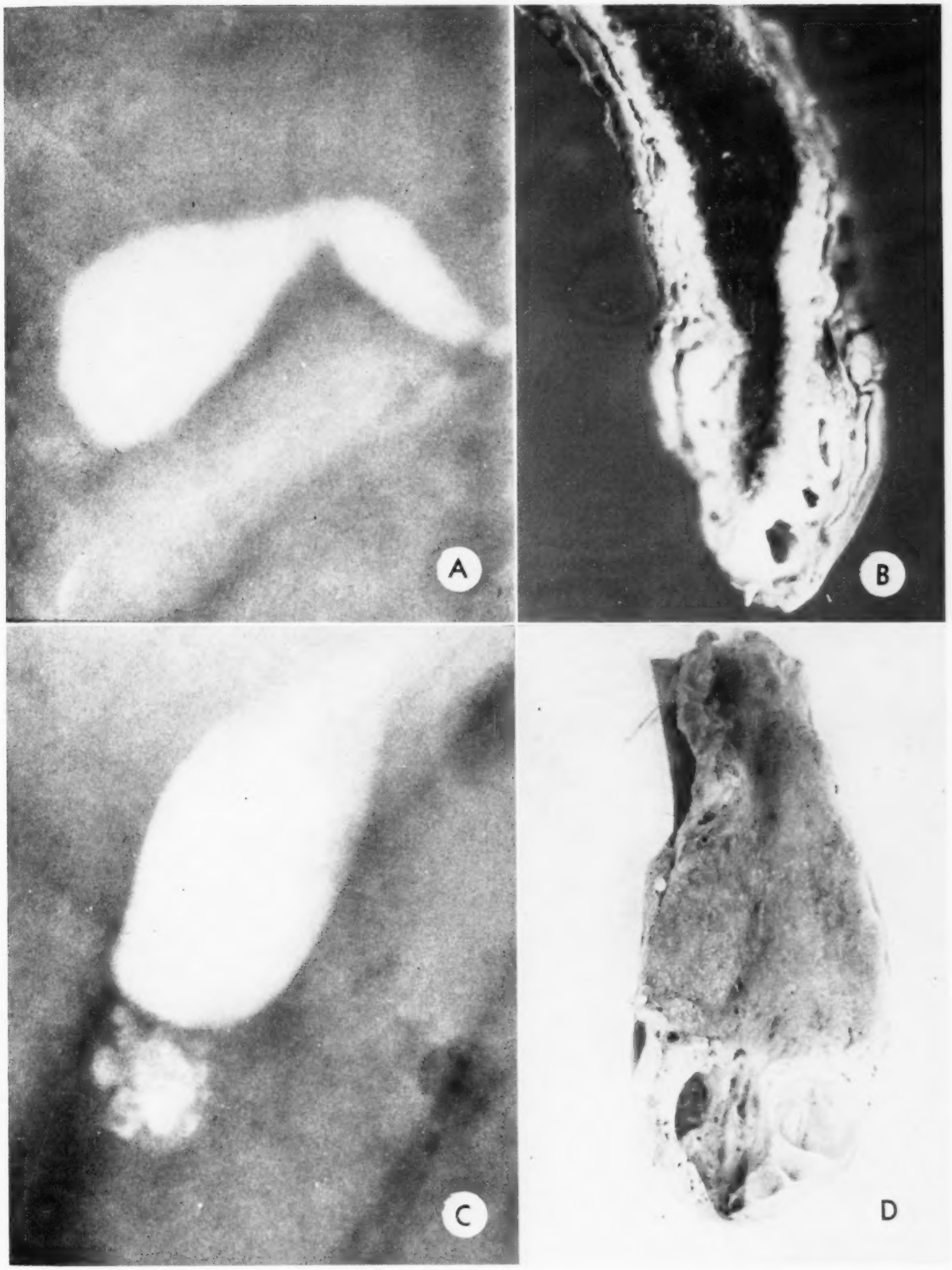

Fig. 25. Adenomyomas with extramural evagination. (*A* and *C*) Cholangiograms. (*B* and *D*) Photographs of the respective operative specimens.

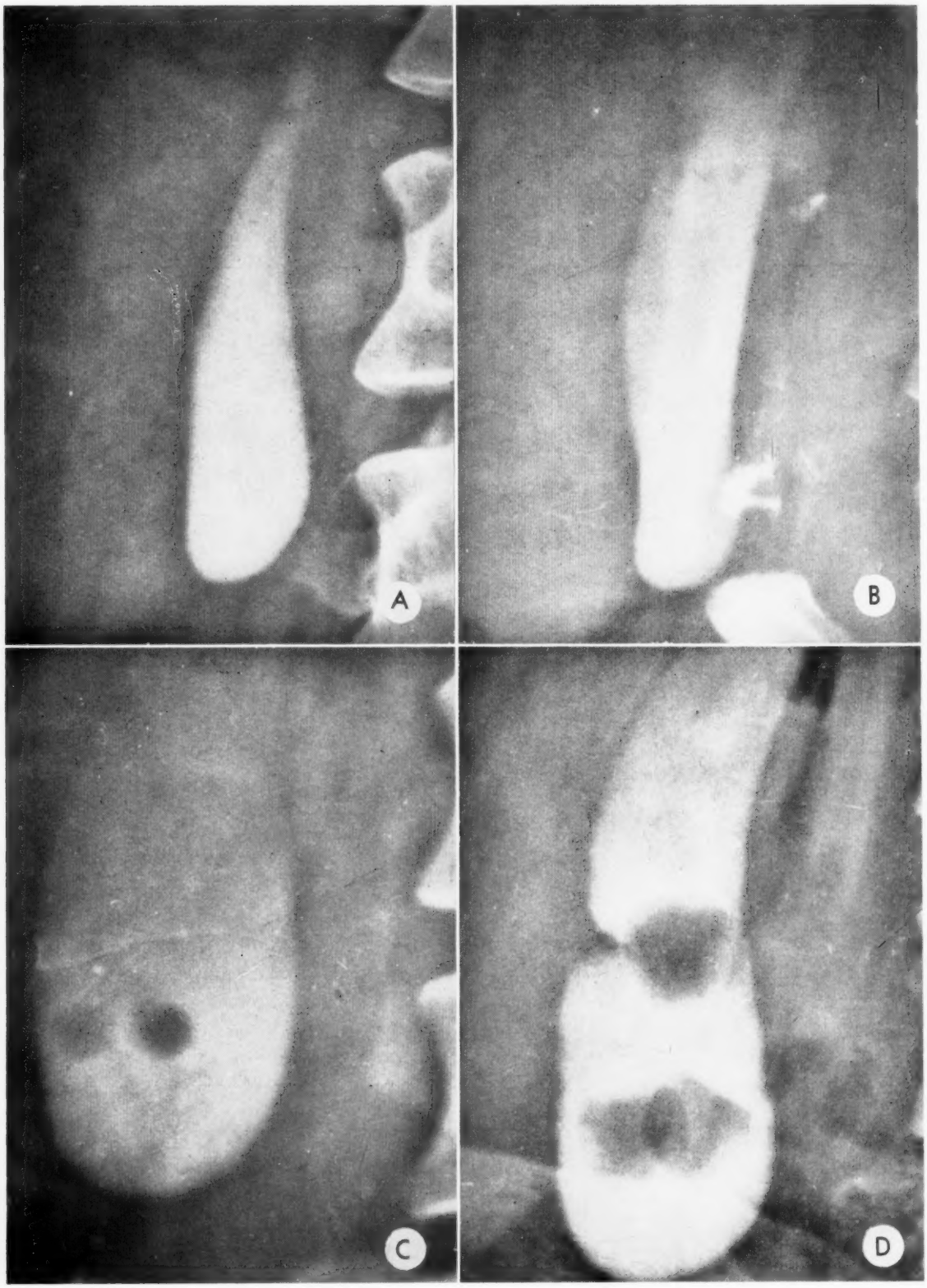

FIG. 26. (*A* and *B*) 1942: Thirty-two year old patient with floating gallstones. (*C*) 1946: Enlargement of gallstones. (*D*) 1953: Acquired type of adenomyomatosis on a fold.

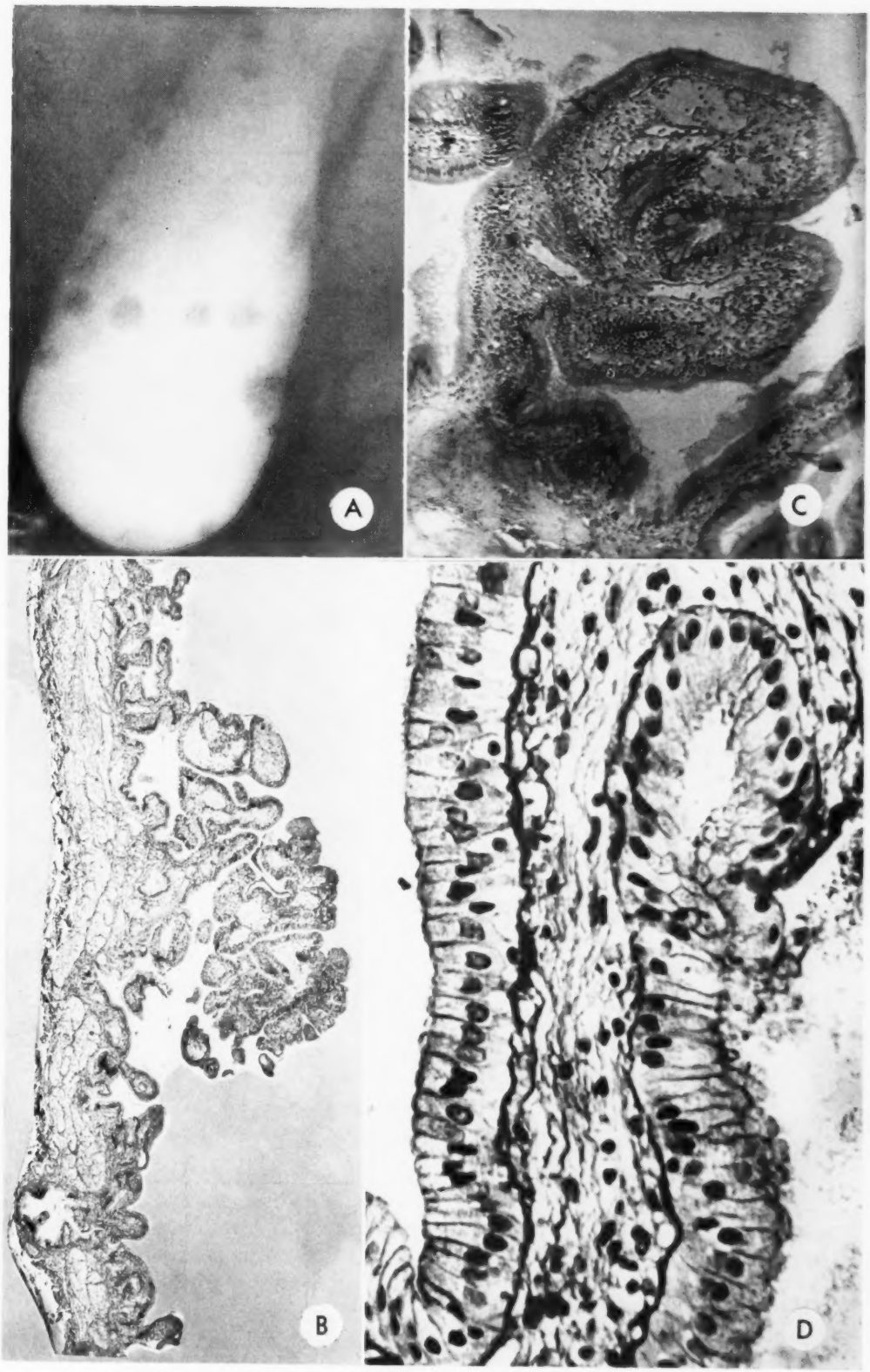

Fig. 27. (*A–D*) Cholesterolosis, four floating calculi and neuromatosis.

TABLE IV

CHOLECYSTOSIS

2,490 PATHOLOGIC REPORTS (1956–1959)

	1956	1957	1958	1959	Total
Cholecystography	5,780	3,936	4,074	4,412	18,202
Cholecystectomy	586	621	668	615	2,490
Total No. of Cases of Cholecystosis	151	274	318	253	996
Per Cent	25.7	44	47.5	41.1	40

Note: The variations in percentage from year to year may be explained by the fluctuations of the pathologists in recording their findings and their desire to choose a more pertinent designation than the commonplace label "chronic cholecystitis."

for study in our 750 bed hospital devoted exclusively to adults is considerable. Annually, there are performed 4,000 to 5,000 cholecystographies, 600 to 650 cholecystectomies and over 300 autopsies (Table IV and V). With so many gallbladders examined by Riopelle himself or his associates, one would think that the problem of neurogenic gallbladders is resolved. Nevertheless, there are many unanswered questions.

The present medical trend is to remove only those gallbladders that contain stones. The prime interest of the radiologist is thus often limited to a search for stones which seem to be the only cause for concern in the clinician's mind.

In the operating theater the gallbladder is opened shortly after its removal in order to extract the precious stones and produce them as evidence to prove that the intervention was justified. Once lacerated and emptied of its content, the specimen provokes little interest on the part of the pathologist who foresees no satisfaction in examining tissues that are in an advanced state of autolysis.

The difficulty of obtaining specimens suitable for careful histologic study and free of stones and also the added work involved in preparing silver and trichrome stains constitute the two main reasons why progress has been slow in chronic diseases of the gallbladder.

In order to assess more accurately the clinical counterpart of these various gallbladder hyperplasias, a larger number of cases is required. These cases should also be free of other types of tissue alteration and be exempt from the repercussions of a pathologic condition in some other organ system. Moynihan,[26] for instance, in his original article on cholesterolosis mentions chronic pancreatitis. Our present conclusions are naturally based on the evidence supplied by patients who have undergone surgery. Severe critics are reluctant to ac-

TABLE V

ADENOMYOMATOSIS

2,490 HISTOPATHOLOGIC REPORTS (1956–1959)

	1956	1957	1958	1959	Total
Cholecystectomy	586	621	668	615	2,490
No. of Cases with Disseminated Form	127	229	261	200	817
No. of Cases with Localized Form (adenomyoma)	1	3	1	6	11
Total No. of Cases of Adenomyomatosis	128	232	262	206	828
Per Cent	21.8	37.3	39.2	33.4	33.3

Note: The above figures refer to specific diagnoses by pathologists. Histologic as well as roentgenologic incidence from written reports is subject to considerable variations depending on interest and opinions of different observers. Minimal forms of intramural diverticulosis are so common in surgically removed gallbladders that busy pathologists may estimate them unworthy of special mention.

cept a comparison of symptomatology before and after cholecystectomy and, moreover, they insist on the necessity of a long follow-up period. The test of time is of course desirable, but cholecystectomy would be carried out infrequently if a required condition for operation were a guarantee that symptoms would not recur.

Objective signs and biochemical tests offer no advantage over the simple clinical history in evaluating follow-up. This clinical appreciation usually reveals that 80 per cent of the operated patients feel much better after surgery. How long will this well-being last? No one can say.

Without siding with interventionists or abstentionists and keeping in mind that the cholecystoses are degenerative processes that increase with age and often remain asymptomatic, we shall sum up our views on the indications for surgery in the following way.

When a gallbladder contains no stones but is hyperfunctional and reveals discrete or overt signs of cholecystosis roentgenologically, with a clinical picture that implicates the gallbladder, the surgeon should use the same discrimination and judgment that he does when faced with a gallbladder containing stones that may or may not be the cause of symptoms.

It is difficult to understand the attitude of certain clinicians who refuse to resort to surgery for these lesions. We know of too many patients who, following a roentgenologic diagnosis of polyps, cholesterolosis or adenomyomatosis, continued to live miserably and were finally relieved by cholecystectomy.

Surgeons could increase our knowledge of gallbladder pathology by observing the following recommendations:

1. Avoid the application of forceps on the fundus of the gallbladder which is the usual site of an adenomyoma.

2. Keep in mind that the lesions described above may not be palpable even when the gallbladder has been emptied by aspiration and the surgeon can roll the gallbladder wall between his fingers.

3. Do not open the freshly removed gallbladder for the simple pleasure of extracting stones.

4. Inject a solution of 10 per cent formalin within the shortest possible time after removal without undue pressure in order to restore the normal shape of the viscus and to prevent the destructive action of bile on the mucosa.

5. Place the formalin-filled gallbladder in a receptacle containing formalin of the same concentration in order to preserve the general morphology of the organ and to permit the pathologist a greater choice of staining techniques.

6. Inform the pathologist of the particular interest in the specimen.

With the great number of cholecystectomies performed daily, such a procedure, if followed faithfully, would elucidate many questions of histopathology of the gallbladder and by the same token permit a better approach to the study of physiologic problems.

Finally, we wish to stress that whether sessile or pedunculated, solitary or multiple these cholesterol polyps are not neoplasms nor is the generalized or localized adenomyomatosis. We believe that the term "papilloma" applied to this lesion by Moynihan,[26] MacCarthy,[22] Kirklin[18] and others is incorrect. We insist on the fact that the polyp in cholesterolosis is not neoplastic contrary to the connotation implied in the term papilloma.

This distinction is fundamental, as Tabah and McNeer[31] have stated, because epithelial proliferations (papilloma, papillary adenoma) possess a strong tendency to malignant transformation. The pathologists of this hospital even observed an example of carcinoma *in situ* in a small true papillary adenoma. Fortunately, papillomas and papillary adenomas of the gallbladder are extremely rare and, when cholangiography reveals a single or multiple small lacunar shadows, the diagnosis of papilloma should be the last choice if only on a statistical basis. This explains our opposition to the indiscriminate use of this

term for all vegetations of the gallbladder wall when it is realized that the true papilloma, an authentic epithelial neoplasm subject to malignant transformation, is extremely rare.

SUMMARY

Hyperplastic cholecystosis designates certain gallbladder conditions that are proliferative and degenerative in nature rather than inflammatory or neoplastic. Consequently, they should be segregated from the miscellaneous group of lesions simply labelled "chronic cholecystitis," because a more precise diagnosis leads to better treatment.

Cholesterolosis, adenomyomatosis and neuromatosis are three forms of hyperplastic cholecystosis of special interest to internists, radiologists and surgeons. They can be currently diagnosed as individual entities by means of selective and serial cholecystography. Since the three conditions are often associated in the same gallbladder, it is important to comprehend their combined influences on cholecystographic results.

Cholesterolosis is characterized by abnormal deposits of cholesterol esters forming yellow specks (strawberry gallbladder), polyps or pseudopapillomas, either solitary or more often multiple.

Adenomyomatosis (also called cholecystitis glandularis proliferans) is defined as a proliferation of surface epithelium with glandular-like formations and outpouchings of the mucosa into or through the thickened muscular layer (intramural diverticulosis).

Neuromatosis indicates proliferation of autonomous neural elements.

A combination of the three above conditions induces the gallbladder to concentrate and expel the contrast medium at too rapid a rate and under excessive pressure. Excess of mucosa is responsible for the hyperconcentration; proliferation of nervous tissue causes hyperstimulation; muscle hypertrophy brings on hyperevacuation. The resulting hyperfunctional syndrome may cause severe and lasting pain, intermittent colics and indigestion, biliary dyspepsia and psychosomatic distress.

Each form of cholecystosis may exist singly or conjointly, with or without stones, with or without mucosal congestion. Roentgenologic features, pathologic correlation, clinical implications and surgical indications are discussed.

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PSEUDOCONCRETIONS IN PRECIPITATE OF CONTRAST MEDIUM IN THE GALLBLADDER*

By GEORG THEANDER
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THE roentgenographic appearance of contrast medium precipitate in the human gallbladder was studied on the basis of observations made in 9 cases.[2] The results showed that stone-like, radiolucent formations may appear within the precipitate despite the definite absence of gallbladder stones, as proved by normal cholecystographic findings, after disappearance of the precipitate. The term, "pseudoconcretions," was introduced to describe such formations, and the importance of their recognition was stressed by the fact that contrast medium precipitate can also contain true concretions. In most of the cases, differential diagnosis was established roentgenographically by observation of the change in shape and subsequent disappearance of the pseudoconcretions contrasted to persistence of the true stones on disappearance of the precipitate. In 1 case, however, radiolucent formations in the precipitate could not be interpreted with certainty before operation.

A case is reported which illustrates the close resemblance between pseudoconcretions and true stones in a precipitate and the consequent difficulty in making a differential diagnosis.

FIG. 1. Cholecystogram with 6 gm. Bi-Na. Stone-like radiolucent formations in gallbladder. Precipitate adherent to fundus in prone position.

REPORT OF A CASE

This female patient, born in 1917, had a history of cerebrospinal syphilis. For several years she had had recurrent upper abdominal pain, probably of biliary origin, but cholecystographic examination in 1956 had shown no abnormality. On re-examination in 1958, after ingestion of 3 gm. bilijodon-natrium (Bi-Na, the sodium salt of iopanoic acid) on each of two consecutive days, a radiopaque mass and several minute, stone-like, radiolucent formations were observed in a normally visualized gallbladder (Fig. 1). On follow-up study within a week, neither the mass nor the gallbladder was demonstrable.

Control cholecystographies, with 3, 6 and 9 gm. Bi-Na, again revealed a radiopaque mass, though less distinctly outlined. The gallbladder and the stone-like formations within it were clearly demonstrable (Fig. 2). After another dose of 6 gm. Bi-Na, a distinct radiopaque mass was demonstrated of approximately the same size as on the first occasion (Fig. 3). It was easily dispersed in the fluid contents of the gallbaldder on change of position but, after an additional dose of 3 gm. Bi-Na, it was again discernible on the following day, as were the radiolucent formations.

Cholecystectomy was performed less than three hours after the last-mentioned examina-

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Fig. 2. Cholecystogram with 9 gm. Bi-Na three weeks later, erect position. Precipitate less distinctly outlined but stone-like formations visualized.

tion. During operation, the cystic duct was ligated as soon as possible to prevent the escape of any of the gallbladder contents but, on roentgenographic examination of the removed gallbladder, no precipitate or stone-like formations were demontrable (Fig. 4). No

Fig. 3. Cholecystogram with 6 gm. Bi-Na on the day preceding operation, prone position. Precipitate adherent to gallbladder wall and stone-like formations visualized.

Fig. 4. Removed gallbladder demonstrating persistence of contrast medium but no precipitate or stones.

stones were found on physicochemical analysis of the gallbladder contents. Contrast medium was present in the specimen (413 mg. per cent iodine) but no precipitate could be obtained, not even by centrifugation.

The pathologist's report was cholesterolosis.

DISCUSSION

The present case of contrast medium precipitate in the gallbladder has several features in common with most of those on record;[1,2] namely, a heavy and radiopaque, dispersible precipitate was produced on cholecystography with the sodium salt of iopanoic acid, and radiolucent formations were demonstrable within it. The phenomenon was reproducible, each time with

occurrence of about the same amount of precipitate. As in the series reported earlier, the patient was a woman with definite gallbladder disease.

In one respect, however, the case under discussion is remarkable. Contrast medium precipitate has not previously been observed to disappear definitively as long as the gallbladder contained sufficient contrast medium to demonstrate the organ roentgenographically. In the present case, the removed gallbladder contained no residue of the precipitate observed in it just prior to operation, although its iodine content was still high.

The radiolucent formations within the precipitate were proved to be pseudoconcretions only by their disappearance from the gallbladder concurrently with the precipitate, in spite of early ligation of the cystic duct during cholecystectomy. Roentgenographically, they had not been observed to change in shape or size; nor had they disappeared before operation as long as the gallbladder was visualized. Moreover, different projections and positions used at cholecystography gave no clue as to their true nature. Re-examination with biligrafin, pheniodol, or some other contrast medium so far not known to produce precipitation, might be conclusive for a differential diagnosis in such cases.

SUMMARY

A case is reported in which a precipitate of contrast medium and stone-like formations within it disappeared from the gallbladder while the concentration of contrast medium in the gallbladder bile was still high. The roentgenographic recognition of pseudoconcretions is discussed.

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THE PROBLEM OF DISAPPEARING GALLSTONES*

ANOTHER CASE OF SPONTANEOUS DISAPPEARANCE ON CHOLECYSTOGRAPHY

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SPONTANEOUS disappearance of gallstones has been considered a great rarity. That 2 cases were encountered at one institution during a period of one year is most unusual and prompts this additional report. The first of these cases, which was proved on October 31, 1958, has been recorded.[11] It concerned a patient, with decisive cholecystographic evidence of gallstones, who at operation was found to have a gallbladder with no residual stones. The second case, encountered in October, 1959, at the Ochsner Clinic, is the subject of this report.

REPORT OF A CASE

A white woman, sixty-seven years of age, came to the Ochsner Clinic on November 12, 1956, for a general physical examination. In 1951, gallstones had been discovered on cholecystographic examination elsewhere (Fig. 1, *A* and *B*). On physical examination no abnormalities were detected except for hemorrhoids and moderate obesity. A functioning gallbladder was demonstrated cholecystographically on November 14, 1956, with numerous radiolucent stones measuring up to 5 mm. in diameter (Fig. 2, *A*, *B* and *C*). Cholecystectomy was advised.

When seen on October 8, 1959, the patient had not had cholecystectomy. At cholecystographic examination on October 13, 1959, a well-visualized and clearly outlined gallbladder was noted. There was no evidence of stones. Many roentgenograms were obtained during a period of six hours, including ones made with the patient in erect and decubitus positions (Fig. 3, *A*, *B* and *C*). A portion of the cystic duct was visible, and it appeared to be normal.

A thorough history was obtained after it was discovered that the stones had disappeared. The patient repeatedly stated that she had been asymptomatic since her visit in 1956. The serum cholesterol level was normal (293 mg./100 ml.).

HISTORICAL REVIEW

It has been recognized for many years that gallstones can pass out of the gallbladder through the biliary ductal system. In 1928, Graham and coworkers[4] stated that "small calculi may pass freely through the ampulla of Vater into the duodenum." Ortmayer and Austin[12] reported 3 instances of recovery of gallstones from the feces of patients who had experienced attacks of biliary colic with jaundice. Gallstones also have been known to erode from the gallbladder into the duodenum or small intestine and produce gallstone ileus.[14]

Since the advent of cholecystography, however, relatively few reports have appeared concerning disappearance of calculi from the gallbladder. In 1937, Rewbridge[13] described 2 cases in which repeated cholecystographic examinations revealed disappearance of radiolucent gallstones, which he attributed to prolonged medical treatment with bile salts and olive oil. These cases were not listed previously in reports of spontaneous disappearance. In 1942, Kommerell and Wolpers[6] recorded a case in which 24 gallstones ranging up to 10 mm. in diameter were seen on serial cholecystograms to become smaller and finally to disappear. Moreau and Moreau[10] reported an instance in which 40 calcified stones 4 to 6 mm. in diameter, which were repeatedly seen in the gallbladder and later were seen in the process of passing through the ducts during episodes of colic, finally disappeared completely.

In 1956, Miller[9] described the case of a twenty-three year old woman who had had

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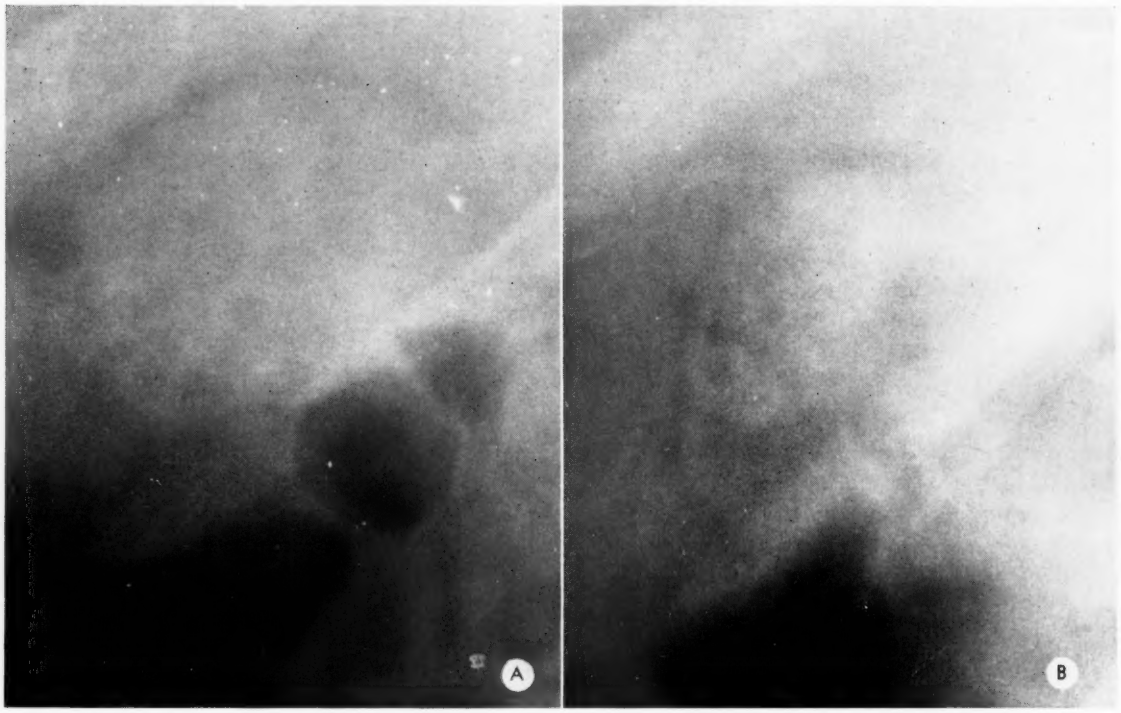

Fig. 1. (*A* and *B*) Cholecystograms made November 24, 1951, show multiple radiolucent gallstones. (We are indebted to Dr. E. Campbell of Memphis, Tennessee for this illustration.)

attacks of biliary colic during pregnancy and in whom numerous small calculi were demonstrated cholecystographically. Although she had had no further episodes of colic, all stones had disappeared from the gallbladder when it was resected seven weeks after the cholecystographic examination. The previously reported case from the Ochsner Clinic[11] resembles in all respects Miller's case. Holt and Hodges[5] men-

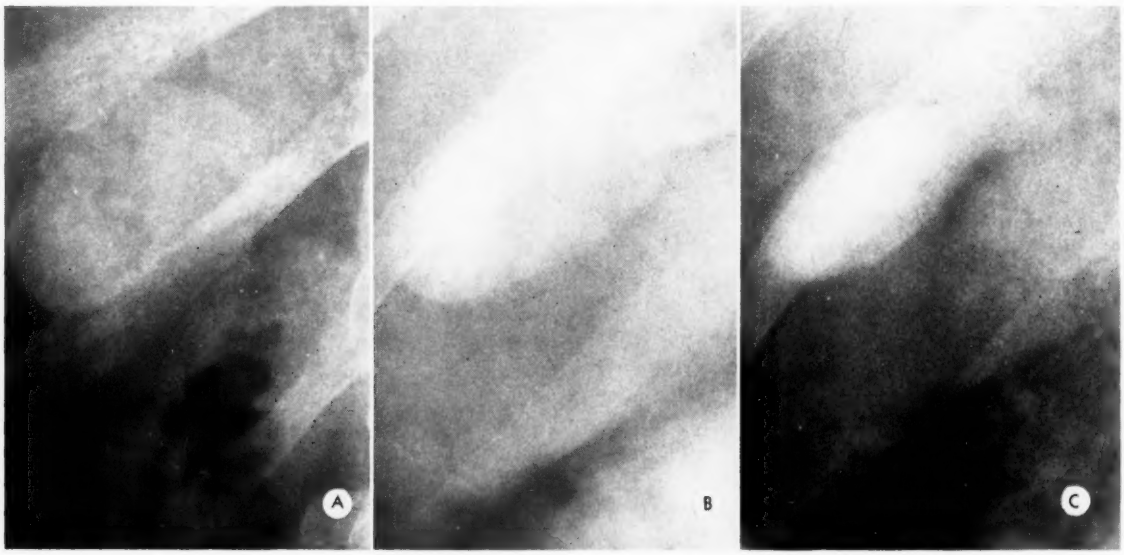

Fig. 2. (*A*, *B* and *C*) Cholecystograms made November 14, 1956, reveal numerous radiolucent gallstones measuring up to 5 mm. in diameter.

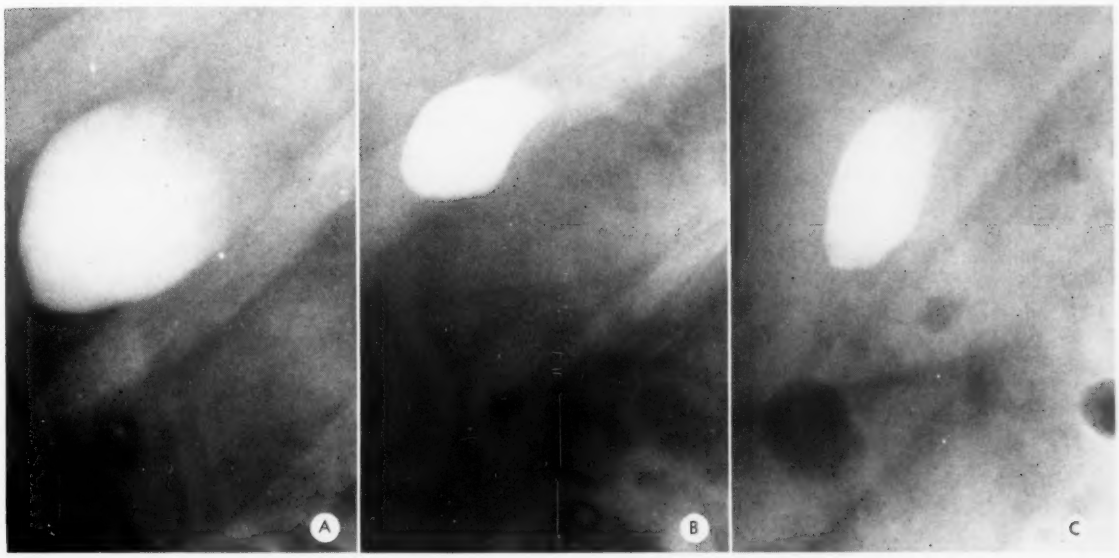

Fig. 3. (*A*, *B* and *C*) Cholecystograms made October 13, 1959, reveal no evidence of gallstones. *C* taken in erect position.

tioned a patient in whom gallstones disappeared after an episode of biliary colic.

More recently, Linsman and Corday[7] described the case of a man, sixty-four years old, in whom 10 radiolucent stones 7 to 9 mm. in diameter were noted on cholecystography in 1954. During the next five years the patient had no episodes of colic, jaundice, or any symptoms that would suggest formation of a biliary intestinal fistula, but in 1958 the stones were no longer apparent on cholescystography. The case herein reported is rather similar to this one.

DISCUSSION

Experience with the spontaneous disappearance of gallstones is limited. The thought occurs, however, that expulsion of calculi from the gallbladder may not be as rare as is generally supposed. In addition to reports of recovery of stones in the feces and their disappearance on cholecystography, other findings may support this concept. Small calculi comprise a limited percentage of roentgenographically visualized gallstones. Since calculi are generally believed to begin as small concretions which grow slowly, one would expect to find a larger percentage of gallstones to be small unless many of them have been spontaneously eliminated. When numerous small stones are visualized, the cholecystograms made after a fatty meal may show calculi squeezed into the neck of the contracted gallbladder or even into the cystic duct. Likewise, during cholecystectomy, surgeons commonly discover stones in the cystic duct or in the common duct, even in the absence of jaundice. McKittrick and Wilson[8] found stones in the common duct of 52 of 100 consecutive patients who had cholecystectomy and choledochostomy for cholelithiasis. Glenn[2] reported stones in the common duct in 7.2 per cent of 2,072 cholecystectomies. In a subsequent review of 5,037 surgically treated patients, Glenn[3] reported that stones in the common duct are almost invariably present in patients with small gallstones and a large cystic duct.

It seems, then, that passage of stones out of the gallbladder may not be as difficult as is usually considered. Cole and Harridge[1] discussed the disappearance of stones from the common duct in 9 patients whose common ducts had been explored, intubated, and later studied by serial postoperative cholangiograms. They believed that some

of the stones dissolved or disintegrated rather than passed through the sphincter of Oddi. Roentgenographic evidence of the disappearance of gallstones is being reported more frequently. There may be other instances of which we are not aware. It seems probable that this may have occurred on some of those occasions when the cholecystographic demonstration of stones was considered incorrect because the surgeon found no stones at operation; the stones may have passed spontaneously. In cases in which spontaneous disappearance of stones is suspected, the radiologist must obtain clear, unequivocal visualization of the gallbladder and as much of the ductal system as possible. Cholecystograms must be interpreted with judicious skill and caution, so that artefacts, superimposed gas shadows, and other possible causes for error do not lead to an incorrect diagnosis.

SUMMARY

Apparent spontaneous disappearance of gallstones is being reported for the seventh time. This is the second case encountered at the Ochsner Clinic in the interval of one year. We believe that gallstones may pass out of the gallbladder more frequently than is generally believed.

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THE DISTENDED GALLBLADDER*

THE VALUE OF A FAT MEAL IN CHOLECYSTOGRAPHY

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THE practice of omitting a fat meal during oral gallbladder examinations is an unwarranted sacrifice of potentially valuable information. It is a deviation from the original technique of examination based on the physiological concept of gallbladder function and the realization that the gallbladder, cystic duct, common duct and sphincter of Oddi behave as a unit. Boyden,[1] Sussman,[16] Ivy[6,7] and others have described the change in gallbladder size after a fat meal and the delicate reciprocal innervation of the gallbladder and sphincter of Oddi. If there is clinical evidence of gastrointestinal disease, the biliary tract cannot be excluded as its source until a complete examination with a fat meal has been performed.

The economic necessity of combining upper gastrointestinal and gallbladder studies in radiology departments where facilities are overtaxed is acknowledged. Unquestionably, a high degree of accuracy has been achieved in recent years in the detection of calculi with and without the visualization of the gallbladder. Because fat meal studies may provide additional information about the gallbladder and in particular about the common bile duct, the present observations are presented.

PHYSIOLOGY

An understanding of normal gallbladder physiology is essential for the performance of a complete roentgen examination of this organ. Boyden and others have shown that within a few minutes after ingestion of a fat meal, gallbladder contractions begin and last for approximately forty-five minutes. These are initiated by the hormonal effect of cholecystokinin produced in the duodenum. During this period the gallbladder contracts 25 to 50 per cent of its original volume. Following an hour to an hour and a half of relaxation, contractions begin again and continue for thirty to forty-five minutes with further reduction in the size of the gallbladder. The gallbladder should be 15 to 20 per cent of its original size at the end of six hours.

Biliary pressures average 35 cm. of water in the intrahepatic bile ducts, 8 to 12 cm. in the common bile duct, and 10 cm. in the gallbladder during relaxation, rising to 25 cm. during contraction. In the presence of dysfunction of the sphincter of Oddi, the pressure may reach 60 cm. of water during gallbladder contraction.

Westphal[17] showed that mild stimulation of the vagus nerve caused the gallbladder to contract and the sphincter of Oddi to relax. Strong vagal stimulation, however, not only increased gallbladder contractions but produced spasm of the sphincter of Oddi with resulting reflux of gallbladder contents into the common hepatic and intrahepatic ducts. Similar observations have been made when the gallbladder had contracted in the presence of spasm of the sphincter of Oddi, either induced by hydrochloric acid in the duodenum or associated with active peptic ulcerations.

NORMAL GALLBLADDER

An adequate study of the biliary tract is one which cannot be left to the judgment of a technician. Examination of the normal gallbladder should include the demon-

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stration of a good concentration of contrast material, absence of calculi, and the partial emptying of contrast material in response to a fat meal. At the beginning of the study, the gallbladder is at the upper limits of normal size or may be distended. Normal size is an arbitrary value depending upon the size of the patient. With the administration of a fat meal and contraction of the gallbladder, the common bile duct may or may not be demonstrable. No great significance is attached to failure of the common duct to visualize unless the suspicions of the radiologist are aroused by clinical indications or by failure of a distended gallbladder to contract.

Once there is a question of possible ductal pathology, every attempt should be made to demonstrate the entire extrahepatic biliary system. This should be done preferably by oral cholecystography with a repeat of the fat meal. If necessary, this should be supplemented by laminagraphy and intravenous cholografin studies.

ROENTGENOGRAPHIC TECHNIQUE

The following technique has been used with minor variations for the past ten years. The supine patient is rotated 20 to 30 degrees, right side down, in order not to obscure the common duct by the spine. Immobilization and compression of soft tissues is achieved by using a pressure binder plus balsa wood or an inflated rubber bag. Exposures should be as short as possible using 75 to 80 kv. and 400 ma. at 0.1 second or 200 ma. at 0.2 second. When the gallbladder is visualized, greater detail may be obtained by the use of a suitable cone and a roentgenogram in the upright position. The use of small cones for detail is of great value and cannot be over emphasized. Sometimes the right posterior oblique as well as decubitus projections are of help.

If these cholecystograms are satisfactory, a synthetic oral fat meal* is given in the department preceded by the subcutaneous injection of 1 cc. 1:4,000 prostigmin. Prostigmin is used for its temporary constriction of the smooth muscle of the sphincter of Oddi, thereby increasing the possibility of visualizing the common duct. Exposures are then made at 8, 15, and 25 minute intervals. If this is not practical, a 10 and 20 or 25 minute study will suffice. By this time, the gallbladder should be about half of its original size and the cystic and common ducts should be visualized in about 75 per cent of patients studied.

In examinations done as part of a routine survey with no symptoms referable to the gallbladder or gastrointestinal tract, a well opacified, contractile gallbladder without calculi is considered satisfactory even if the common duct is not visualized. In all other instances the fat meal is repeated in an attempt to make the gallbladder contract and to visualize the common duct. If this is unsuccessful, intravenous cholografin is employed.

With the use of cholografin, the biliary ducts start to visualize within 10 to 15 minutes after injection. The common bile duct is best seen at about 30 minutes since the density of the shadow will have decreased at 45 minutes. The gallbladder, on the other hand, is best demonstrated at 2 to 2½ hours. Patients rest in the waiting room or return to their rooms during this interval.

Since contrast material in the duodenum will obscure adequate visualization of the distal common duct, there should be no hesitation in supplementing this examination with laminagraphy. The common duct is best demonstrated in the right anterior oblique projection with cuts between 12 and 14 cm. In taking laminagrams of the average patient, *delay of more than 30 minutes after injection will result in decreased clarity with the diminishing concentration of the contrast material.*

Although such a study as just outlined is time consuming for a routine examination it may prove rewarding by revealing an unsuspected neoplasm of the pancreas before jaundice supervenes. Inflammatory disease

* The synthetic fat meal "Ediol" used in these studies was supplied by Schenley.

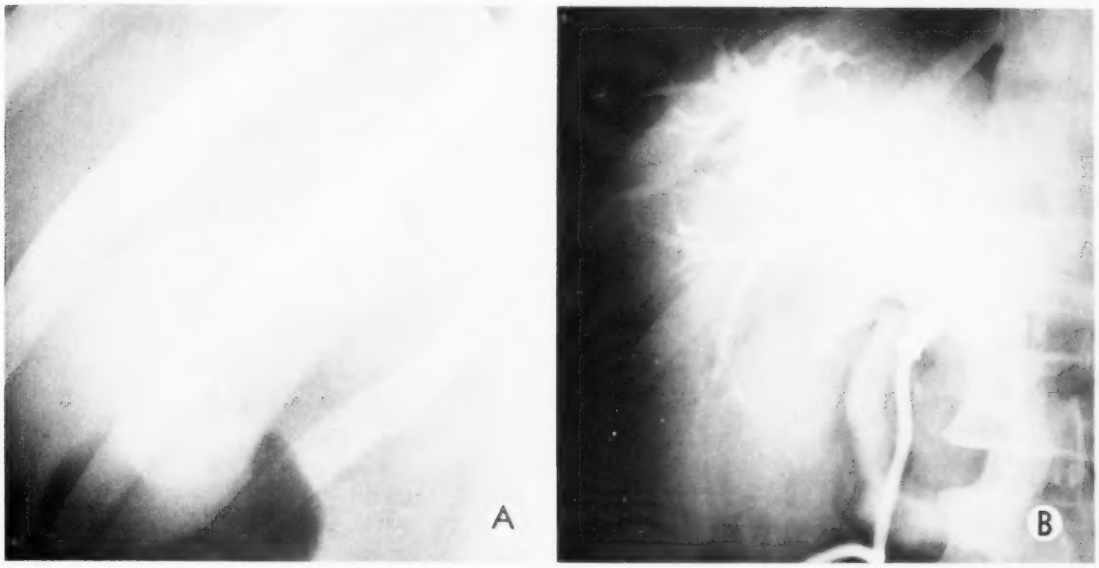

FIG. 1. W. L., a forty-six year old Negro male, was admitted because of increasing epigastric distress of six weeks' duration. Pain occurred about fifteen minutes after eating. Alcoholic intake had been reduced because of pain. Patient had noted a 30 pound weight loss. Admission hemogram, liver function tests and gastrointestinal roentgen studies were normal. Serum amylase on admission was very high but the bilirubin was normal. Oral and intravenous roentgen studies of the biliary tract revealed a good concentration of contrast medium in a distended gallbladder which failed to respond to a combination of fat meals and antispasmodics. (*A*) Cholecystogram shows a distended gallbladder. The common duct was never demonstrated. Five weeks later the patient became jaundiced with an additional 10 pound weight loss and recurrence of pain. An abdominal roentgenogram showed persistent retention of the original contrast medium in the gallbladder. Operation revealed carcinoma of the head of the pancreas with lymph node metastases. (*B*) Operative cholangiogram shows obstruction of the pancreatic portion of the common duct with proximal dilatation. A by-pass procedure was performed and the patient was discharged.

of the pancreas, dysfunction of the sphincter of Oddi and unsuspected calculi in the common bile duct can also be recognized preoperatively and suitable therapy planned.

DISCUSSION

The visualization of a well filled or distended gallbladder that fails to empty in response to a fat meal should alert the radiologist to the possibility of urgent and often serious pathology involving the bile ducts. The concentration of contrast material may vary somewhat with the amount of the agent used and the condition of the gallbladder. However, it can be an indication of liver damage even without clinical evidence of jaundice. Nonvisualization of the gallbladder or a delay in concentration of the contrast medium may occur with liver disease or after a recent pancreatitis.

When the gallbladder fails to decrease in size, adequate visualization of the common bile duct will usually reveal the location and, at times, the nature of the disorder (Fig. 1, *A* and *B*). The diagnostic possibilities are limited for practical purposes to inflammatory and neoplastic changes involving the common duct, sphincter of Oddi and duodenum in the region of the sphincter of Oddi. Cancer of the common duct or head of the pancreas and chronic pancreatitis involving the head of the pancreas all may produce partial and at times complete obstruction and distortion of the pancreatic portion of the common duct (Fig. 2, *A* and *B*, Fig. 3, *A–D*, and Fig. 4, *A–D*). It day be impossible roentgenographically to differentiate early carcinoma of the head of the pancreas from pancreatitis and surgery is then imperative. With the pathology

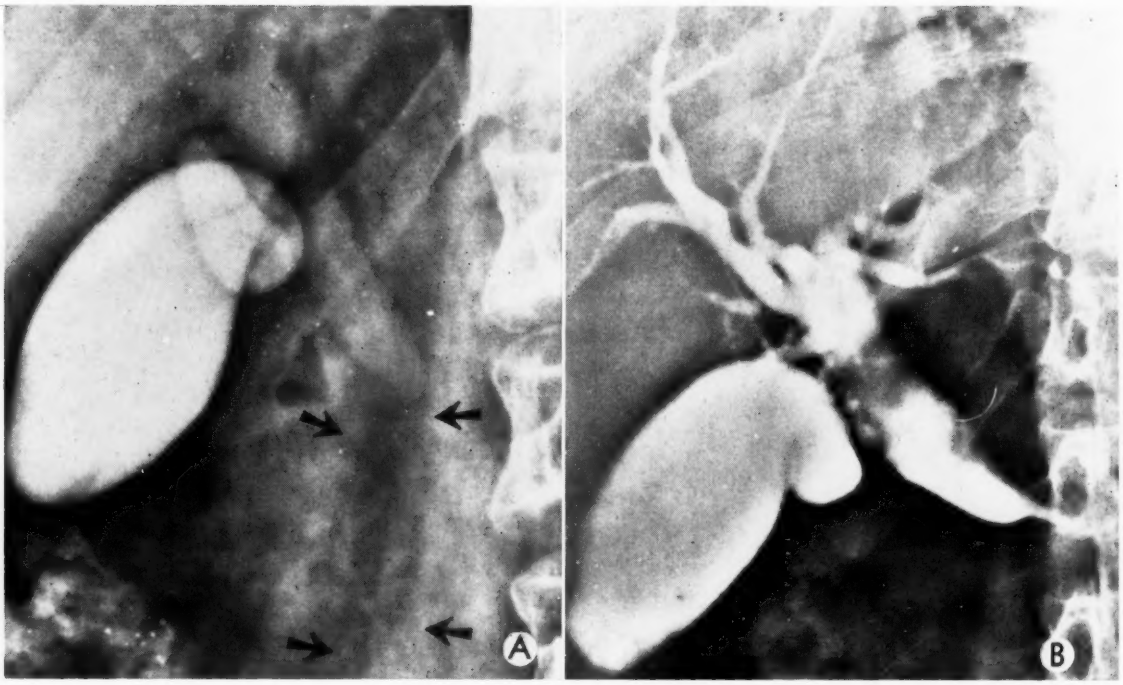

FIG. 2. C. J. M., a sixty-two year old white male, diabetic for four years, complained of inconstant dull mid-abdominal pain and 30 pound weight loss within two months. Stools were occasionally frothy. Positive physical findings were limited to a murmur of mitral stenosis. Alkaline phosphatase, bilirubin and amylase were normal. Injection of morphine produced epigsatric pain for two and one-half hours followed by vomiting. (*A*) Cholecystogram shows an average-sized gallbladder without calculi and with no response to a fat meal. The proximal common duct is dilated and appears to end in an abrupt taper. The distal common duct (first seen after logEtronic reproduction) is extremely narrow and deviated laterally. Patient refused operation and was discharged but was readmitted with jaundice two weeks later. Operation revealed carcinoma of the pancreas with liver metastases. (*B*) Operative cholangiogram shows complete obstruction and dilatation of the proximal common duct. A by-pass procedure was performed.

located at the lower end of the duct, proximal dilatation increases as the condition progresses to complete obstruction.

During an abdominal laparotomy for disease unrelated to the biliary tract, the discovery of an abnormally distended gallbladder should not be ignored, particularly if the gallbladder canot be emptied by squeezing, and if the common duct is dilated. If it is impractical to determine the nature of the obstruction at the time of the operation, it should be investigated roentgenographically as soon as possible. Waiting until the occurrence of jaundice will usually make curative surgery for neoplastic obstruction too late and that for nonmalignant obstruction much more difficult.

Lesions which cause obstruction in the region of the sphincter of Oddi include carcinoma of the terminal bile duct arising in the wall of the ampulla of Vater and prolonged spasm of the sphincter of Oddi which may progress to hypertrophy and fibrosis (Fig. 5, *A* and *B*.). Of these lesions, in most instances only the carcinoma goes on to complete obstruction. Dilatation of the common duct ending in an abrupt taper at the sphincter of Oddi requires surgical exploration even though the well filled gallbladder contains no calculi to account for the recurrent attacks of biliary colic. Carcinoma of the duodenum in the vicinity of the sphincter of Oddi may produce obstruction of the terminal bile duct with proximal dilatation of the common duct but rarely proceeds to complete obstruction.

Delay in reaching an accurate diagnosis until occurrence of clinical jaundice may

Fig. 3. F. H. S., a fifty-seven year old white male, was admitted for the seventh time, with hemoptysis and hematemesis of one week's duration. A subtotal gastrectomy had been done six years previously for duodenal ulcer. The patient complained of recurrent postoperative right upper quadrant pain coming on an hour after meals and becoming more severe six days prior to admission. Admission hemogram, liver function studies, and serum amylase were normal. Upper gastrointestinal roentgen studies failed to show the source of the bleeding. (*A*) Oral cholecystogram shows an average-sized gallbladder with no response to a fat meal. (*B*) Laminagram demonstrates dilated cystic duct and part of a markedly narrowed common duct. Compare with *C*, a normal cholecystogram made seven years previously. (*D*) Operative cholangiogram confirms the marked narrowing and lateral displacement of the distal common duct with proximal dilatation. A by-pass procedure was performed and the patient was discharged after a complete recovery.

FIG. 4. J. B., a thirty-nine year old white male, had numerous admissions to several hospitals over an eight year period for severe epigastric pain radiating to the back. Pain was relieved only by narcotics. Edematous

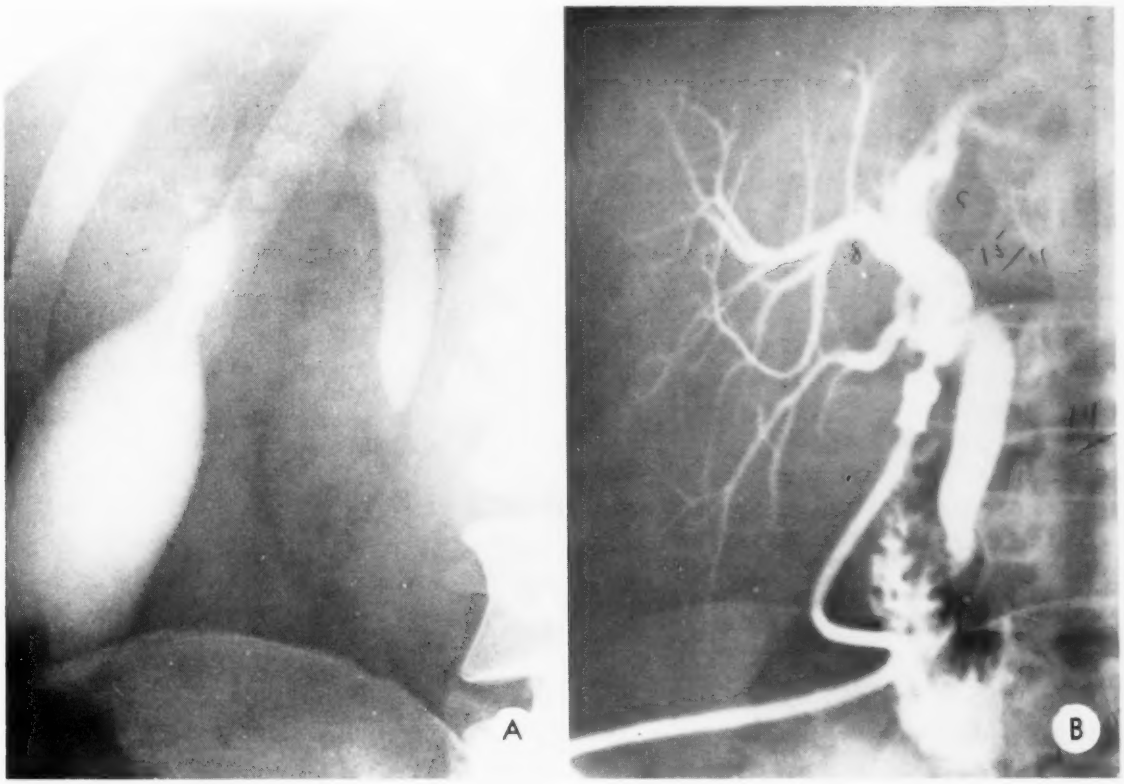

Fig. 5. C. K., a fifty-two year old white male, had a two and one-half year history of intermittent abdominal pain. Attacks of pain were severe, radiating to the flanks, and persisting from three hours to four days. Nausea and vomiting were also associated with the pain for which, occasionally, a hypodermic injection was necessary. His family stated that at times he looked "yellow." During the two and one-half years there was a loss of 45 pounds. Gastrointestinal studies were normal. (*A*) Cholecystogram made after a fat meal reveals a distended gallbladder and common duct dilatation, tapering at the distal end. (*B*) Operative cholangiogram shows marked narrowing of the distal common duct with proximal dilatation. The narrowed common duct segment was due to fibrosis of the sphincter of Oddi, for which a sphincterotomy was done.[13]

make roentgen diagnosis impossible and curative surgery too late. Obviously, as emphasized above, surgical intervention may be too late even before the appearance of jaundice, but the earliest possible diagnosis should be the objective. Jaundice frequently introduces the possibility of hepatocellular disease with the additional delay and increased liver damage involved. Surgery may be postponed because of this

and chronic recurrent pancreatitis had been demonstrated at previous operations. The patient was cachetic and had persistently elevated serum amylase values. (*A*) Oral cholecystogram made following a fat meal shows narrowing of the pancreatic portion of the common duct with proximal dilatation and reflux into the intrahepatic ducts. (*B*) Operative cholangiogram made fourteen months later shows increased dilatation of the proximal common duct with persistent distal narrowing. A by-pass procedure was performed, using the gallbladder, and the patient discharged. He was readmitted ten months later with back pain and 20 pound weight loss. Serum amylase again was elevated. (*C* and *D*) Cholografin studies with laminagraphy show occlusion of the pancreatic portion of the common duct, pancreatic calcification and a functioning cholecystojejunostomy. At re-operation a dilated, sacculated pancreatic duct containing numerous calculi was opened and anastomosed to a Roux loop of jejunum.

diagnostic dilemma until liver function is not adequate to withstand the stress of radical surgery.

SUMMARY

The practice of omitting a fat meal during cholecystography sacrifices valuable information, particularly about the common bile duct. Normally, the gallbladder contracts 25 to 50 per cent of its original volume during the first 45 minutes after a fat meal. Visualization of the common duct is increased by the subcutaneous injection of 1 cc. 1:4,000 prostigmin before this meal. If the gallbladder is distended and fails to contract, visualization of the common duct by repeat fat meal or by intravenous cholografin studies supplemented with laminagraphy will usually reveal the location of the pathology.

Lesions which prevent the gallbladder from emptying are limited to inflammatory and neoplastic changes involving the common duct, sphincter of Oddi, and duodenum in the region of the sphincter of Oddi. These include cancer of the common duct or head of the pancreas, chronic pancreatitis in the head of the pancreas, prolonged spasm or fibrotic stenosis of the sphincter of Oddi, and carcinoma of the duodenum in the vicinity of the sphincter of Oddi.

Delay in reaching a diagnosis until the occurrence of clinical jaundice makes preoperative roentgen diagnosis virtually impossible and may make curative surgery too late.

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CHOLECYSTOGRAPHY WITH ORABILEX*

A COMPARATIVE STUDY

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DURING the past year a number of comparative reports have been written on cholecystography. After the usual experimental studies on toxicity and the lethal dose in animals, it was found that bunamiodyl (orabilex) could be given in a much larger amount than some of the iodides now in use in cholecystography. Although, in tests on dogs, about 70 per cent of the orabilex was excreted by the kidneys, its prolonged use caused no renal damage.

Orabilex has been described by several authors as a white, odorless, slightly hygroscopic powder that contains 57 per cent iodine. It is interesting to compare the physical data of this medium with those of the two more recent compounds in use: orabilex, molecular weight 661 and 57 per cent iodine; telepaque, molecular weight 571 and 66.7 per cent iodine; and priodax, molecular weight 494 and 51.5 per cent iodine. Since the usual dose of telepaque is 3 gm. and of orabilex is 4.5 gm., there is more iodine given with the latter drug. This is of advantage if the increased amount of iodine produces no increase in unfavorable reactions.

In the report by Whitehouse and Fink[8] on 1,000 cases evaluated with telepaque and orabilex, a smaller dose of telepaque (2 gm.) was used than by most clinics. They noted about 10 per cent fewer severe side effects and stressed the decreased residue in the colon.

Teplick *et al.*[5] used a dose of 6 gm. orabilex and 3 gm. telepaque. When they changed to 4.5 gm. orabilex, the side effects remained essentially the same.

Bickham *et al.*[2] reported 350 patients with similar findings. They repeated the examination in 40 patients who had nonvisualized or poorly visualized gallbladders by giving a double dose, 9 gm. orabilex. The side effects showed little change; only 1 of 26 previously nonvisualized gallbladders showed faint visualization and 9 of the 14 poorly visualized gallbladders were slightly improved. This gives an accuracy of about 98 per cent on the initial examination and makes the double dose repeat examination unnecessary.

We repeated cholecystography in 10 patients, using orabilex after there had been nonvisualization with telepaque, and 5 showed a fair degree of visualization. This same percentage was found when telepaque was used as the repeat medium. When cholecystography is repeated on the following day, a larger amount of iodide may be reabsorbed from the intestine and a comparative study would not be as accurate as if a week transpired between examinations.

CLINICAL STUDY

A trial study to determine the effectiveness of orabilex was made of 300 routine cases coming for examination at random. A similar study for purposes of comparison was made of 299 patients using 3 gm. doses of telepaque. No attempt was made to regulate the dose of either medium according to body weight. It was definitely established that 3 gm. telepaque or 4.5 gm. orabilex is more than is needed for the small patient. No patient had clinical jaundice. Ambulatory outpatients were given orabilex and inpatients were given telepaque. Part of the group had blind study, the radiologist not knowing which medium was used. The usual preliminary preparation was carried out in all instances. The patients, were instructed to eat a regular evening meal and to take 6 capsules at 5 minute intervals, without chewing, with

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Orabilex was supplied by E. Fougera and Company; telepaque was supplied by the Winthrop Chemical Company.

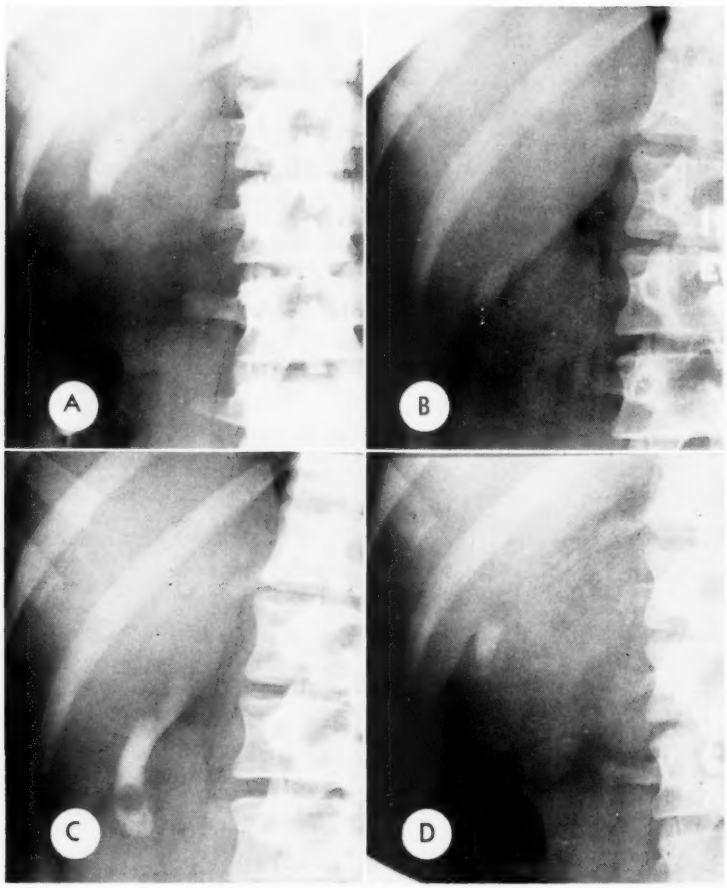

Fig. 1. (*A* and *B*) Gallbladder demonstrated with telepaque. (*C* and *D*) Same as *A* and *B*, two days later using orabilex as the contrast medium. Some of the increase in density may be due to reabsorption of iodide over the forty-eight hour period.

fruit juice or a small amount of water. Nothing else by mouth was allowed after 10 P.M. At 9 A.M. the following day, when the patient reported for examinations, a routine form was filled out with questions regarding symptoms following ingestion of the 6 capsules. After the conventional posteroanterior and lateral decubitus roentgenograms were made, the fat meal was administered and additional roentgenograms were made from thirty minutes to one hour. It was found that a *post cibum* study about thirty minutes after the fat meal and in deep Valsalva breathing very frequently produced good visualization of the cystic and common ducts. The degree of opacification was evaluated in order to compare both media (Fig. 1, A–*D*).

REACTIONS

The patients' reactions after ingestion of orabilex and telepaque are tabulated in Table I. In the orabilex group, there were 4 patients who complained of dysuria and 3 had a moderate skin reaction. One of

Table I

REACTIONS

	Orabilex	Telepaque
	(Per cent)	(Per cent)
Nausea	10.5	9.5
Vomiting	1.2	1.6
Intestinal Cramps	8.2	9.9
Dysuria	1.3	1.5
Diarrhea	—	10.0
Skin Reaction	1.0	0.9

these 3 developed hives; she was found to be moderately hypertensive and with an allergic response to several tests, but not to iodine. Two patients complained of dizziness, but these patients were hypertensive and on the rice diet treatment. Five patients complained of nausea and vomiting. In the telepaque group there were 4 cases with the same symptoms. All of our patients in both groups were sick with some type of complaint and this may account for more severe reactions than those described by others.[1,4,6,7] As a whole the reactions were slightly less with orabilex.

RESULTS

In comparing the two most commonly used contrast media, orabilex and telepaque, the controversial factors were studied.

1. *Visualization of the Gallbladder.* Routine forms were used as supplied by E. Fougera & Company. As shown in Table II, 78.6 per cent using orabilex showed good visualization against 66 per cent with telepaque—an increase of 12.6 per cent. In the nonvisualized group, telepaque gave a higher percentage.

Table II

GALLBLADDER VISUALIZATION

	Orabilex	Telepaque
	(Per cent)	(Per cent)
Good	78.6	66.0
Fair	11.2	13.2
Poor	5.6	6.6*
None	4.2	9.9*

* These were pathologic gallbladders.

2. *Demonstration of Calculi.* There were a few more calculi demonstrated with orabilex than with telepaque, but the variation was within normal limits (Table III). Ten cases showed nonfilling with telepaque; of these 5 showed normal filling with orabilex, 2 showed poor filling and 3 showed nonfilling. The 5 were considered normal.

3. *Duct Filling.* There was a much higher percentage of duct visualization with orabilex than with telepaque and this may be attributed to a more complete absorption of the medium (Table IV). The ducts are often visualized by taking the *post cibum* roentgenograms in the Valsalva maneuver.

Table III

DEMONSTRATION OF CALCULI

	Orabilex	Telepaque
	(Per cent)	(Per cent)
Opaque Stones	2.2	3.9
Nonopaque Stones	9.3	8.5

Table IV

DUCT FILLING

	Orabilex	Telepaque
	(Per cent)	(Per cent)
Cystic Duct	34.0	15.6
Common Duct	14.5	5.1

4. *Colon residue.* One of the most pertinent findings in the use of orabilex was the more complete absorption and the lessening of colonic residue (Table V). This often made the routine tap water enema unnecessary.

Table V

COLON RESIDUE

	Orabilex	Telepaque
	(Per cent)	(Per cent)
Moderate	8.6	16.5
Slight	8.9	9.9

CONCLUSIONS

The reactions are slightly less, using orabilex, although 1 case had a rather severe allergic reaction.

There is a significant increase in duct visualization with orabilex, especially with the Valsalva maneuver.

There is definitely less colonic residue with orabilex.

There is still a place for further develop-

ment of gallbladder media until one is found which will be 100 per cent effective.

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GAS EMBOLIZATION OF THE PORTAL VENOUS SYSTEM*

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IN THE interpretation of roentgenograms of the acute abdomen, the right upper quadrant is routinely scanned for evidence of gas in the gallbladder and/or biliary tree. If such gas is visualized and there are multiple, distended, continuous loops of small intestine, the diagnosis of gallstone ileus is given most serious consideration. It is often difficult to distinguish air in the gallbladder from air in the duodenal loop or in the hepatic flexure of the colon. However, gas in the common duct and the hepatic radicles is, in most cases, readily distinguishable from enteric gas by its roentgen appearance. The pencil-like gas shadow in the common duct and in the hepatic ducts, continuing into branching hepatic radicles, is characteristic.

This case report calls attention to an apparently similar gas pattern in the liver seen on roentgenograms of the acute abdomen which, however, is indicative of an entirely different pathologic condition. This pattern is due to gas embolization of the portal venous radicles as a result of a breakdown of the endothelial barriers between the small mesenteric veins and the gas in the enteric lumen.

Wolfe and Evans[7] reported 6 cases of gas emboli in the portal radicles occurring in infants. They reasoned that gas under increased tension in the lumen of the bowel somehow gains entrance to the mesenteric veins and collects in the portal system. The accompanying clinical conditions of their cases were: erythroblastosis fetalis (3 cases), congenital intestinal obstruction followed by surgery (2 cases), and necrotizing enteritis with accompanying diarrhea (1 case). In their review of the literature, Wolfe and Evans were unable to find any reference to this condition up to 1952, and we have found no reference subsequent to their report. Furthermore, no case of gas embolization of the portal venous system in an adult has been found in our perusal of the literature.

In the case to be presented, an elderly male developed mesenteric thrombosis. There was necrosis and dissolution of natural barriers which allowed the enteric gas to enter either one or many mesenteric veins and to be transported as bubbles which became arrested in the portal radicles.

REPORT OF A CASE

S. O., a seventy-seven year old white male, was admitted to the Jewish Hospital on September 21, 1953, with the chief complaint of confusion and headache of several days' duration. The patient was a known hypertensive who, during the preceding week, had become progressively more confused. On the day of admission, he had diarrhea and fecal incontinence.

Physical and laboratory findings. Examination on admission showed an elderly, thin, uncooperative male who complained whenever he was moved. He was confused and disoriented. Physical examination revealed a temperature of 100° F. (rectal) and a blood pressure of 180/88. The pulse was 76 and irregular. The brachial vessels were hard and tortuous. The heart was enlarged to the left. Abdominal examination was negative except for bilateral inguinal herniae. Rectal examination revealed bright red blood on the tip of the examining finger. The left pupil was larger than the right. The remainder of the neurologic examination was negative. Examination of the blood showed a hemoglobin of 14.5 gm., and a white blood cell count of 19,600, with a differential count of 14 per cent stabs, 80 per cent polymorphonuclears and 6 per cent lymphocytes. The urinalysis showed 1+ albumin. On the day following admission, the blood pressure had fallen to 140/70. An electrocardiogram showed auricular fibrillation with rapid ventricular response and

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FIG. 1. Upright roentgenogram of the abdomen showing many markedly distended loops of small intestine with long fluid levels. The fluid in the same loop is at the same height, indicative of an adynamic ileus. The branching pattern of air in the portal venous system is seen distributed peripherally in the liver area (Fig. 2*A*).

frequent ventricular extrasystoles. The abdomen had become rigid and tender. The inguinal herniae were readily reducible. No bowel sounds were heard. Aspiration of stomach contents revealed coffee ground material.

Roentgen findings and diagnosis. Examination of the abdomen showed many markedly distended loops of small intestine (Fig. 1). Each loop contained a large amount of gas with considerable fluid. The fluid in the same loop was at the same height presenting a long air-fluid level indicative of adynamic ileus. A branching pattern of air was noted in the hepatic area on the roentgenograms. Assuming that this air was in the biliary radicles, the presence of a cholecystoenteric fistula was suggested. The possibility of mesenteric arterial occlusion secondary to embolization had been considered clinically, since the patient was fibrillating. Influenced by the finding of air in the liver together with the distended small bowel loops, the clinical diagnosis was changed to gallstone ileus.

Surgical findings. An exploratory laparotomy, which was performed twenty-four hours after admission, showed extensive gangrene of the small intestine as a result of a superior mesenteric artery thrombosis. There was foul smelling exudate within the peritoneal cavity and palpation revealed the branches of the superior mesenteric artery to be cord-like and pulseless, with the exception of one branch to the most proximal jejunum. Many air bubbles were seen in the various branches of the superior mesenteric vein. When the mesentery was transilluminated, the flow of gas bubbles in the mesenteric veins was described by the surgeon as "resembling the bubbles of gas seen in certain neon light signs." An extensive small bowel resection was performed and the proximal jejunum anastomosed to the terminal ileum. The patient's postoperative course was rapidly downhill and he died twenty hours after surgery. Permission for a postmortem examination was refused.

Pathologic findings. The specimen consisted of a portion of small intestine, which was 7 feet in length, and the attached mesentery. Over the major portion of the specimen, the intestinal wall was dark brown and the lumen was filled with reddish brown hemorrhagic material. The vessel within the mesentery appeared, grossly, to be thrombosed. No crepitus was noted. Microscopic examination showed the mucosa to be denuded, and an intense polymorphonuclear leukocytic infiltration was present in the submucosa and superficial muscle layers. Large numbers of bacteria were present in the necrotic debris of the mucosa. The arteries within the mesentery were extremely arteriosclerotic, and the veins were greatly dilated. No thrombi were encountered. No cystic spaces suggesting gas filled blebs were seen in the sections. The diagnosis was infarct of the small intestine.

DISCUSSION

In the case presented, there was necrosis and dissolution of natural barriers, which probably allowed the enteric gas to enter either one or many mesenteric veins and to be transported as bubbles and become arrested in the portal radicles. As these bubbles of gas became arrested in the portal bed, they formed a continuous pattern which was visualized against the homogeneous background of the liver parenchyma.

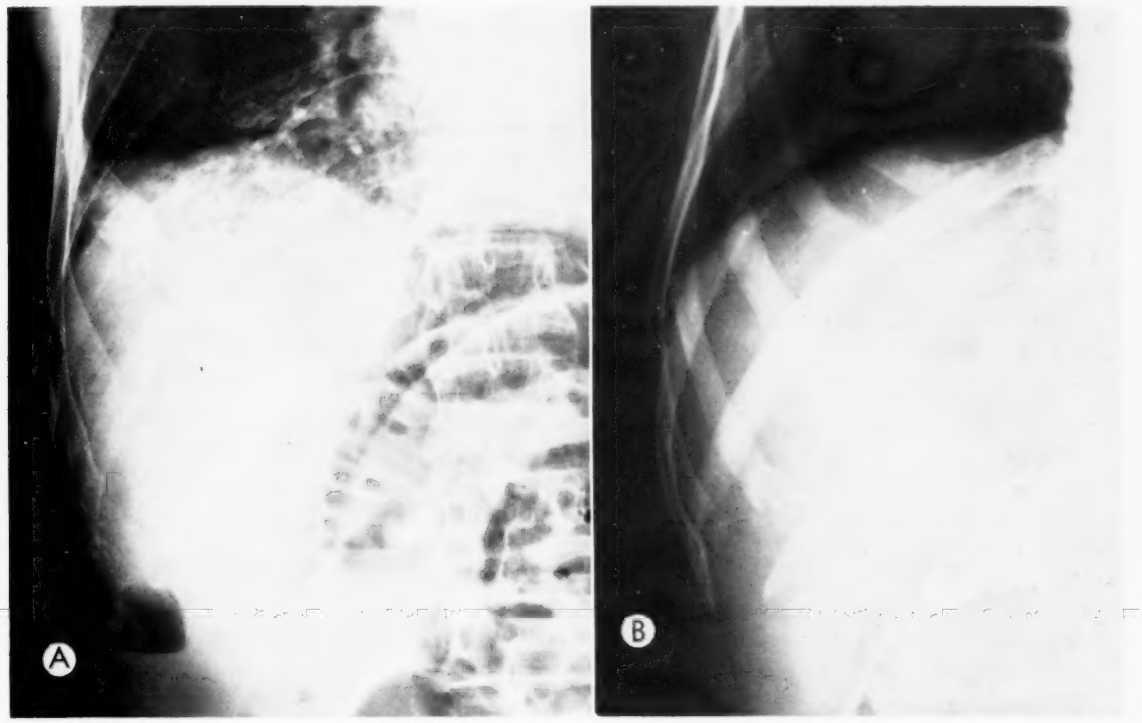

Fig. 2. (*A*) Detail of the liver area of the upright roentgenogram of the abdomen in Fig. 1, showing the distribution of gas in the finer peripheral venous radicles in the liver caused by the centrifugal flow of the portal venous blood. (*B*) Detail of the liver area in a case of fistulous communication between the gastrointestinal and biliary tracts. Note the characteristic branching pattern of gas in the major biliary radicles resulting from the continuous centripetal flow of the secreted bile.

Air in the portal veins is indicative of an altogether different clinical entity than is air in the biliary tree. Therefore, it is important to attempt to differentiate these two, similar, branching patterns. Air in the biliary tree is due to a fistulous communication between the intestine and the gallbladder or common duct. In this condition, gas enters the major radicles and is prevented from entering the finer radicles by the continuous centripetal flow of the secreted bile. By contrast, in gas embolization of the portal venous system, the bubbles are carried into the finer peripheral venous radicles in the liver by the centrifugal flow of portal venous blood. Thus, these two patterns of gas in the liver, each characteristic of a different pathologic entity, can be roentgenographically differentiated (Fig. 2, *A* and *B*) by noting whether the peripheral fine radicles are involved.

In the case described, no gas blebs were seen either grossly or microscopically in the intestinal wall, but gas bubbles were visualized being transported in the veins draining the same area. There being no source of gas in the intestinal wall, the immediate source of gas in the veins was almost certainly intraluminal gas under increased tension in distended ischemic small bowel loops. This was probably also the case in the infant with necrotizing gastroenteritis reported by Wolfe and Evans.[7] The gas blebs of pneumatosis intestinalis may have been the immediate source in 3 of the cases in infants presented by Wolfe and Evans, but, here again, the most probable indirect source was intraluminal gas.

Another possible source of gas in the portal veins is interstitial gas generated locally by the fermentation of invading bacteria such as *Clostridium* or *B. coli*. Cases with such intramural gas collections due to clostridium infections have been reported

by Kellert and Meeler[4] and Boggs *et al.*[1] In these cases, the collections were small and multifocal, and, therefore, if a fistulous connection develops with a vein, only the small focal collection is available for embolization. By contrast, in pneumatosis intestinalis, the individual collections of gas are often much larger and probably interconnected.

The possibility also exists that a large focal collection of gas under increased pressure can be generated by gas forming bacteria in an abscess originating in an area being drained by the portal venous bed. This gas may subsequently enter the portal channels through a break in the endothelium. If the gas were a relatively insoluble one, such as methane, it could, *per se*, form an embolus of sufficient time duration to be visualized. If the gases produced were extremely soluble, such as CO_2 and H_2S, they, *per se*, could not cause a significant embolus. However, even though the origin of the gas bleb were the generated CO_2 and/or H_2S, the blebs, from inception, would be found to contain both nitrogen and oxygen which must diffuse into the blebs until their partial pressures in the bleb equilibrate with their tissue tensions. These secondary gases, because of their insolubility, could give rise to significant embolization. Gazin *et al.*,[3] Nitch[5] and Plenge[6] have stated that the presence of oxygen in a tissue bleb excludes the possibility of bacterial origin, since no bacterium is known to produce oxygen. We believe these opinions to be incorrect, as oxygen would be present due to gas diffusion from the blood and tissue fluids.

Regardless of the mechanism of passage of gas into the portal radicles whereby several clinical conditions may thus manifest themselves in children, in our case, and in several unreported instances noted by Felson,[2] gas in the portal veins in adults has been a manifestation of mesenteric thrombosis.

An experiment was performed on a dog to determine the amount of gas necessary to visualize the portal bed. As little as 5 cc. of air injected slowly into a small mesenteric vein was sufficient to permit visualization of that portion of the portal bed occupying the left lobe of the liver. Although no measurements of venous pressure were done in this experiment, it might be predicted that, if there were sufficient air to fill the entire portal system in a retrograde manner, an acute portal hypertension would be produced. This may be one of the factors which caused significant intraluminal hemorrhage in the patient reported, as this finding is much more common with venous than with arterial thrombosis.

SUMMARY

1. The roentgenographic examination of the abdomen of a patient with an acute superior mesenteric arterial occlusion showed gas emboli in the portal venous bed.

2. Gas emboli in the portal vein and gas in the biliary duct system can be differentiated on roentgenograms.

3. The possible sources of gas emboli in the portal vein radicles are discussed.

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DUODENOCOLIC APPOSITION*

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BY DUODENOCOLIC apposition is meant a constant, closer than normal, positional relationship between the descending duodenum and the transverse colon.

In 1914 Harris[9] called attention to a previously undescribed condition; namely, severe constriction of the second portion of the duodenum where it is crossed by the transverse colon. In 6 surgically verified cases he demonstrated an abnormal peritoneal fold or band (a remnant of an embryologic structure—the anterior mesogastrium) extending variously from the liver or gallbladder region to and across the duodenum to the transverse colon or transverse mesocolon.

This is in line with the opinion of Caffey,[3] who believes that compressing peritoneal bands are due to failure of resorption of fetal peritoneal folds.

McIntosh and Donovan[11] state that the regularity with which bands and adhesions occur in conjunction with anomalies of intestinal rotation has been emphasized by virtually all students of the problem. Their incidental nature was fully established by Frazer and Robbins,[6] who proved conclusively that such bands were the result, rather than the cause of anomalies of rotation. They also demonstrated that certain constant and well established areas of thickening in the parietes of the peritoneal cavity are the normal residua of the process of intestinal rotation.

ANATOMIC CONSIDERATIONS

According to Gray's Anatomy, the descending duodenum is crossed in its middle by the transverse colon, the posterior surface of which as well as the anterior surface of the crossed duodenum are uncovered by peritoneum (Fig. 1). There is a small quantity of connective tissue between them. Anteriorly, the supracolic and infracolic portions of the duodenum are covered by peritoneum, but not posteriorly.

FIG. 1. Anatomic drawing illustrating the normal relationship between the transverse colon and the descending duodenum. From: Anatomy of the Human Body by Gray, 25th Ed., Lea & Febiger, Philadelphia, 1948.

The lower end of the foregut (that is, the duodenum at the level of the biliary papilla) is, at a very early period, a fixed and constant point.

At first the hindgut occupies the midline of the abdomen. From its upper end a retention band, a mesenchymal thickening in the mesentery, passes upwards to the condensed tissue about the origin of the superior mesenteric artery. This retention band does not keep pace in growth with the gut or mesentery, so that it forms an anchorage of the artery as growth proceeds. The gut, being "hitched up" by the band, is bent by

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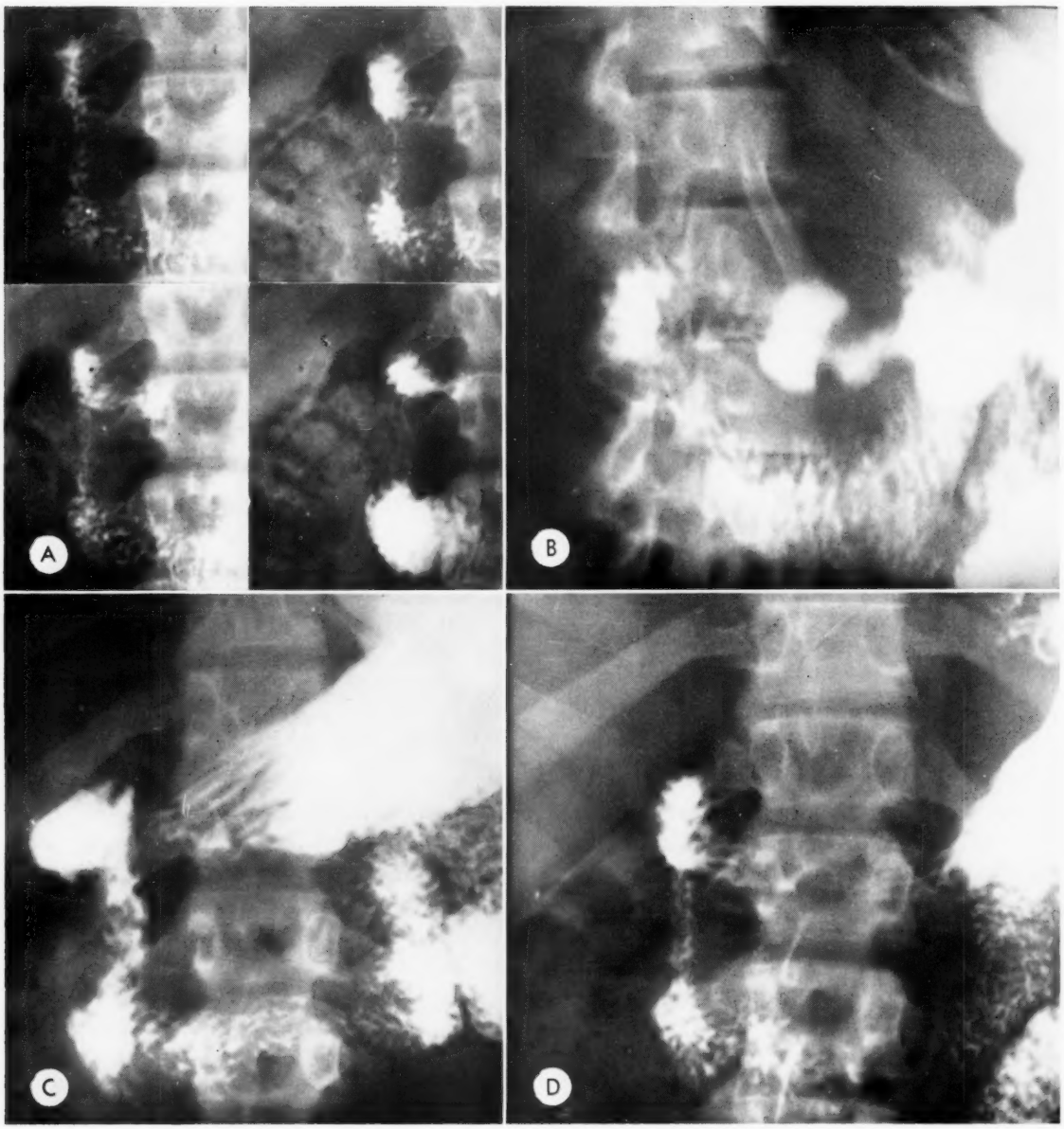

FIG. 2. (*A*, *B*, *C* and *D*) Note greater than normal duodenocolic apposition on various roentgenograms of one patient. There is mutual indenture between the descending duodenum and the transverse colon with varying degrees of stasis in both the descending duodenum and the transverse colon.

it at the junction of midgut and hindgut (proximal third of the transverse colon). This flexure is known as the colic angle which is, at a very early period, a fixed point at the upper end of the hindgut.

The duodenum and the colic angle are close to each other, and, being fixed points, the growth of the embryo causes their relative approximation, so that they form a narrow isthmus (the duodeno-colic isthmus) to which the two limbs of the midgut loop are attached.

The midgut grows very rapidly in length and forms a large loop, convex forwards. The rapid growth of the midgut loop and of the liver so diminishes the available ab-

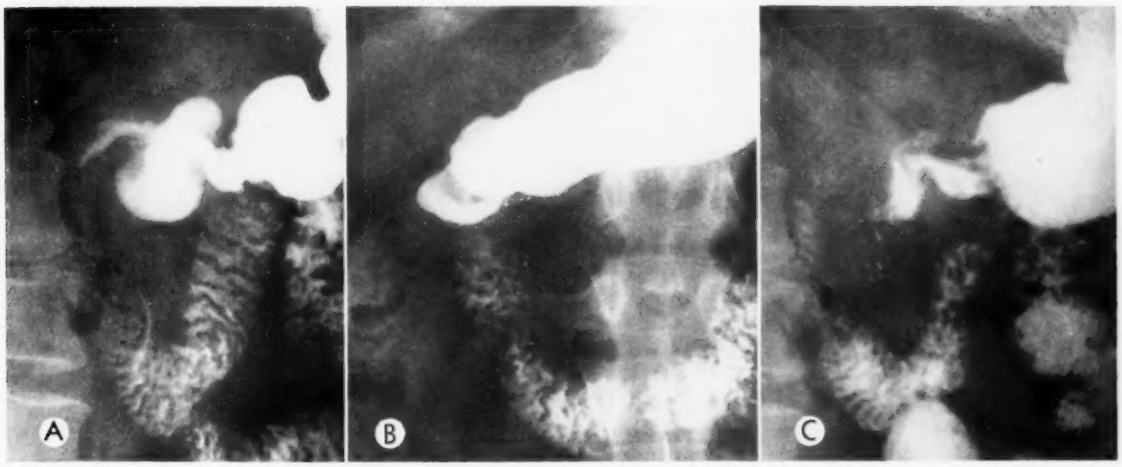

Fig. 3. (*A*, *B* and *C*) Duodenocolic apposition with the same features as described in Figure 2.

dominal space that the former is extruded into the root of the cord as a temporary and physiologic umbilical hernia.

Upon the return of the now highly coiled intestine into the abdomen, the characteristic rotation, already begun at the time of herniation into the cord, is completed. In this process the cecal end of the colon is

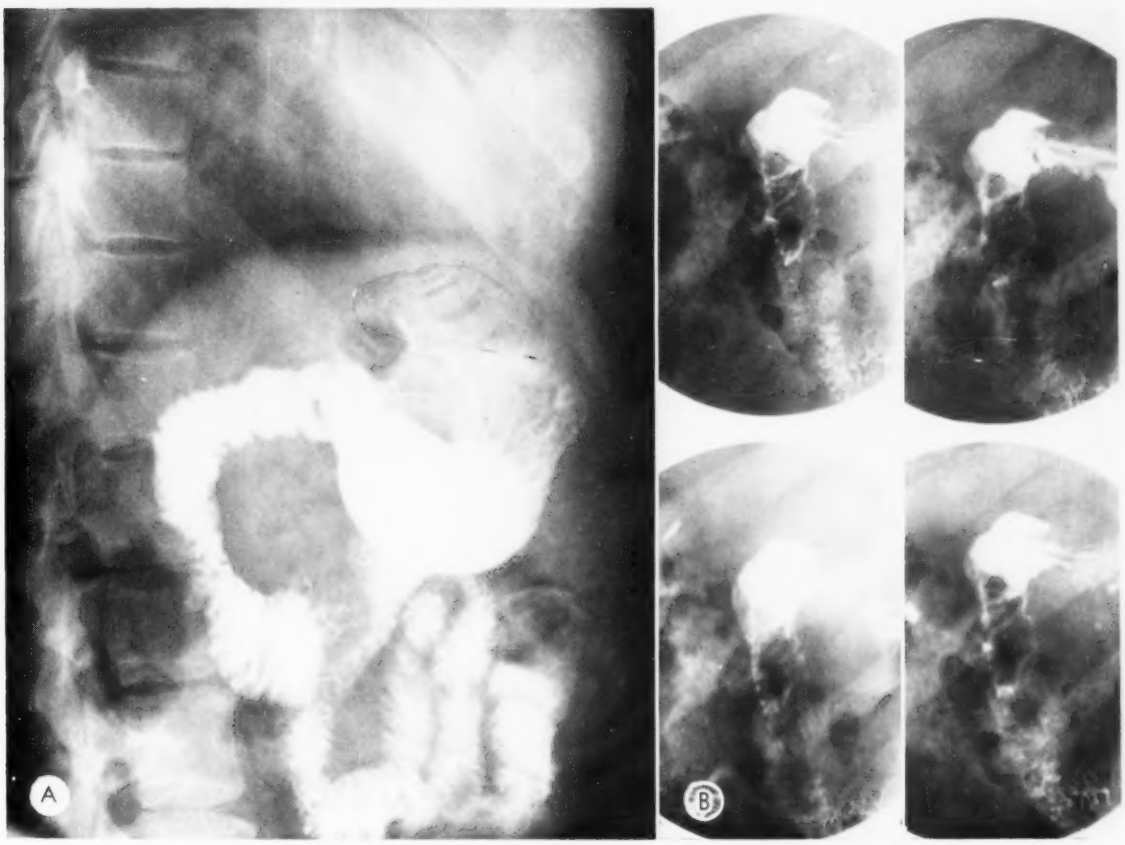

Fig. 4. (*A* and *B*) Duodenocolic apposition with the same features as described in Figure 2.

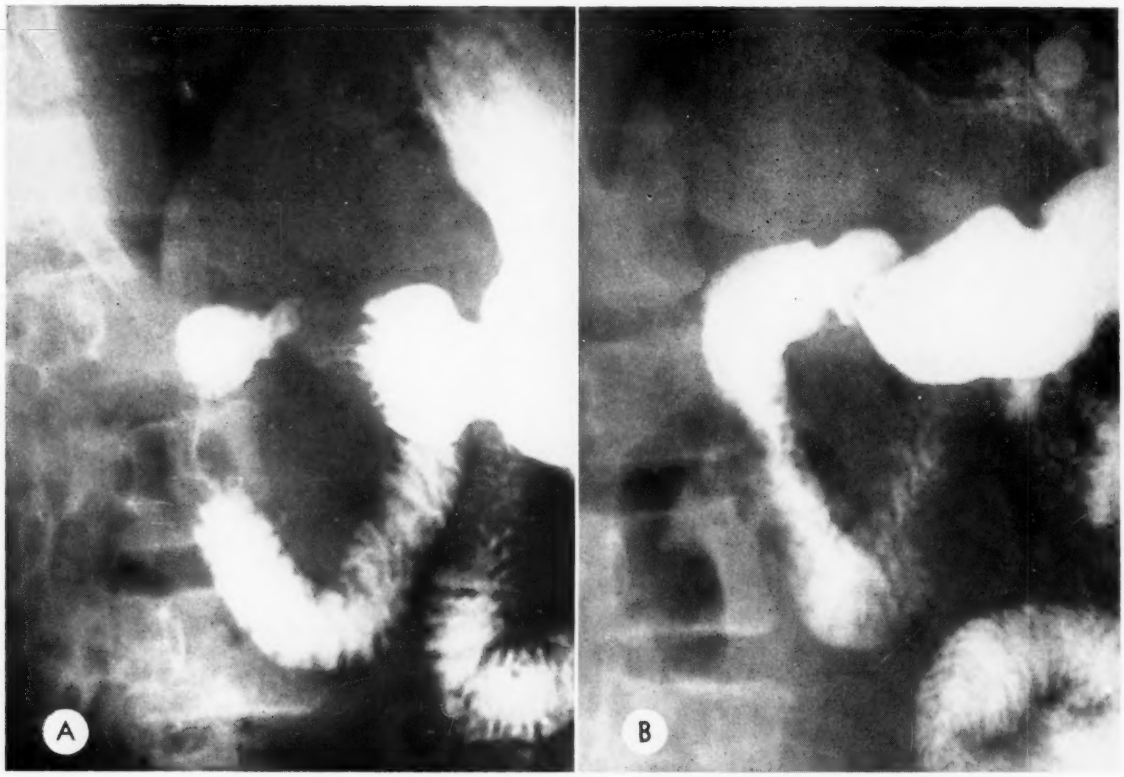

FIG. 5. (*A* and *B*) Duodenocolic apposition with the same features as described in Figure 2.

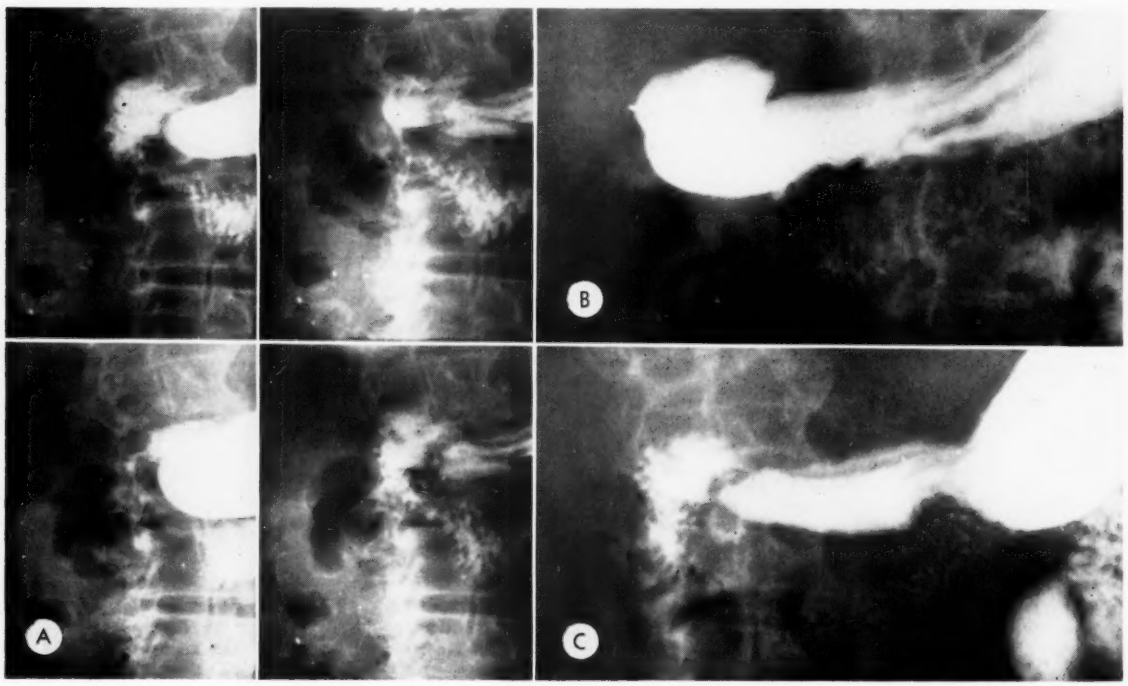

FIG. 6. (*A*, *B* and *C*) Duodenocolic apposition with the same features as described in Figure 2.

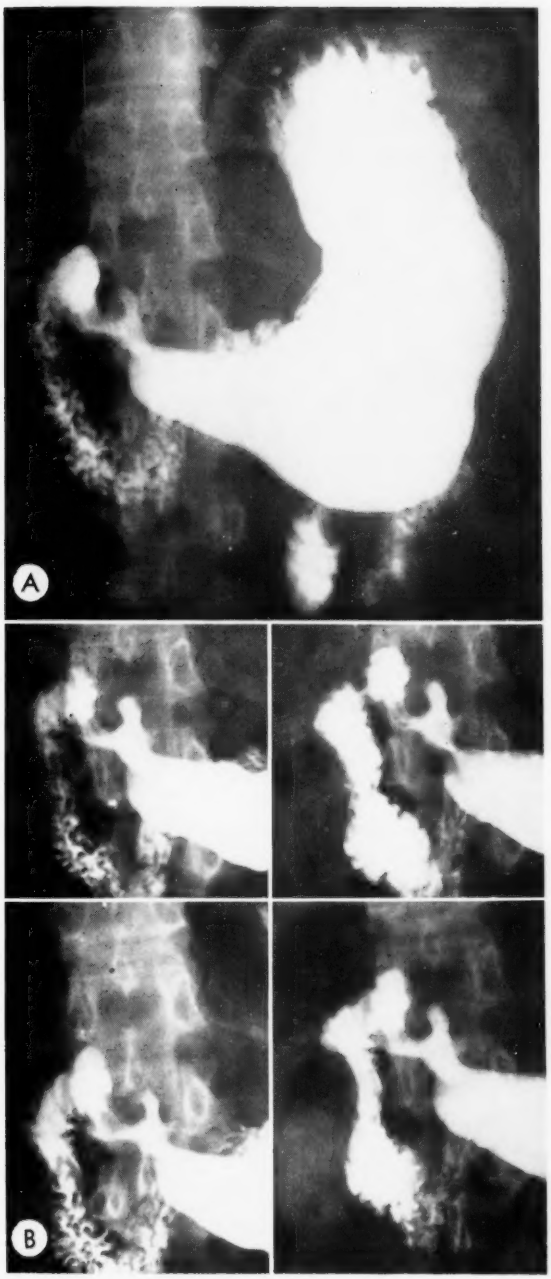

Fig. 7. (*A* and *B*) Duodenocolic apposition with the same features as described in Figure 2.

carried over to the right, whereby the forward transverse colon crosses ventrad to the duodenum and the small intestine lies mostly at the left of the cecum.

During early development there is an anterior as well as a posterior mesentery. The posterior mesentery extends throughout the entire length of the intestinal canal, while the anterior one extends caudad only so far as the proximal third of the duodenum. That portion of the anterior mesentery which is to lie caudad of the diaphragm is called the ventral mesogastrium.

Normally, that portion of the anterior mesogastrium which extends from the duodenum to the liver becomes the hepatoduodenal ligament while the caudal edge of the anterior mesogastrium at its junction with the duodenum fades out and becomes imperceptibly lost in the epithelium covering this portion of the bowel.

Occasionally, the transverse colon, as it crosses the duodenum in its evolution, becomes attached to the caudal edge of the mesocolon and draws it out in a fold more or less distinct which persists and forms what is called the hepatocolic ligament. This consists of a peritoneal fold and extends usually from the gallbladder or the cystic duct across to the transverse colon or the transverse mesocolon, but does not affect the duodenum.

More rarely, we find the ventral layer of the caudal edge of the anterior mesogastrium persisting or existing as a distinct fold and extending from the liver or gallbladder across the ventral surface of the duodenum and becoming lost in the peritoneum over the pancreas or in the ventral layer of the transverse mesocolon. It is the presence of such an abnormal fold of the ventral layer of the caudal edge of the anterior mesogastrium that caused the constriction described in Harris's cases and which in its mildest form may be the etiology of duodenocolic apposition. In the absence of a definite fold but in the presence of some thickening between the two, the opinion of McIntosh and Donovan[11] may prevail—that some areas of thickening in the parietes of the peritoneal cavity are the normal residua of the process of intestinal rotation.

There is a great variability in the degree of obstructive force exerted by this band or ligament on the duodenum with a conse-

quent wide range in the intensity and nature of the symptoms produced. The severe cases are recognized at or near birth, but the very mild ones, the ones with only a gentle mutual indenture between the duodenum and the transverse colon, escape notice. These are the ones in which we are currently interested.

ROENTGEN FINDINGS

Roentgenologically, in slightly over 3 per cent of 500 duodenal ulcer patients, there was noted a rather constant, closer than normal, apposition of the transverse colon to the descending duodenum sufficient to produce some degree of mutual indentation (Fig. 2–7). This could be easily appreciated when both were simultaneously opacified. Whatever the explanation, the roentgen manifestation was clear—closer than normal constant duodenocolic apposition. The implication was also clear—some degree of duodenal stasis and its role in the physiopathology of the duodenal bulb. There was additionally some degree of stasis in the transverse colon as demonstrated by the partially widened bowel containing an unusual amount of gas and feces.

SUMMARY

The anatomic relationship between the descending duodenum and the transverse colon is discussed and the significance of duodenocolic apposition considered.

The roentgen findings in duodenocolic apposition are described and illustrated by typical roentgenograms.

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Vol. 83, No. 5

SUBMUCOSAL HEMATOMA OF THE DUODENUM: DISCUSSION AND REPORT OF A CASE

By WILLIAM E. KIRKPATRICK, M.D.
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SUBMUCOSAL hematoma of the duodenum is a very rare lesion. Perry and Shaw,[13] in reporting the results of duodenal involvement in 17,652 autopsies found only one case. Watanabe and Inouye[19] reviewed the medical literature in 1956 and found less than a dozen cases reported. The condition is usually due to blunt trauma. Most of the cases reported were either found at autopsy or on surgical exploration of the abdomen. The preoperative diagnosis is infrequently made. In 1954, Felson and Levin[2] reported 4 cases of the lesion and, in one of these cases, they made the first preoperative diagnosis that had been made by means of an upper gastrointestinal series and presented criteria for the diagnosis of the lesion roentgenologically. Since their report, there have been 4 additional cases published[8,14,15,19] in which the diagnosis was made roentgenologically. Three of the cases diagnosed correctly roentgenologically were treated conservatively and, while the operative diagnosis was not established, the patients became asymptomatic and ensuing examinations showed that the lesions had disappeared. The clinical and roentgenographic evidence offered in the cases is thought to be conclusive for the diagnosis. In the other two cases,[14,19] the roentgenologic diagnosis was confirmed surgically. The case reported here was diagnosed preoperatively and surgical exploration verified the presence of the lesion.

REPORT OF A CASE

A.M.A., a twenty-nine year old white female, was admitted to Montgomery County Hospital, Conroe, Texas, on September 12, 1959. She had been involved in an automobile accident the previous night and had been examined and dismissed from the emergency room of a hospital in Houston, Texas, following the accident. She later developed chest and back pain which led to hospitalization. The history included the fact that she had struck the steering wheel in the accident. Physical examination at the time of admission was entirely negative except for contusions, which were mainly confined to the chest and right side of the body. Laboratory studies were all within normal limits except for an elevated white blood cell count with a shift to the left.

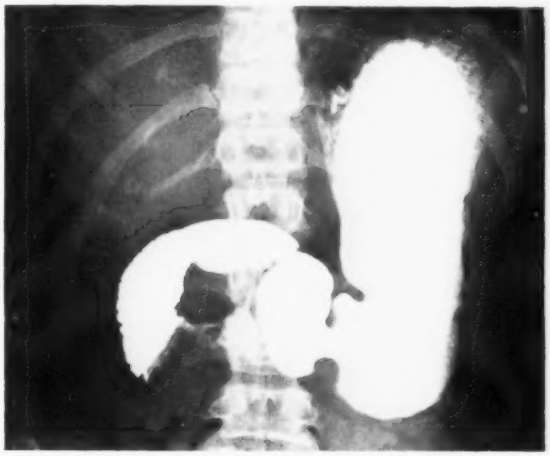

FIG. 1. Roentgenogram showing an obstructive lesion of the duodenum.

On September 13, 1959, the patient began to vomit and to complain of pain in the upper abdomen and of nausea. Skull roentgenograms on September 14, 1959 were negative. Nausea, vomiting, and abdominal pain continued and became worse. On September 17, 1959, an examination of the upper gastrointestinal tract showed an intramural lesion beginning near the junction of the second and third portions of the duodenum which crowded together the valvulae conniventes in the third portion of the duodenum and almost completely obstructed the duodenum. The stomach and duodenum proximal to the lesion were dilated (Fig. 1). There was a tender mass palpable in the epigastrium. Roentgenograms at two and twenty-four hours following ingestion of the barium showed that only a small amount had passed the mass. A roentgenologic diagnosis of a submucosal hematoma was made.

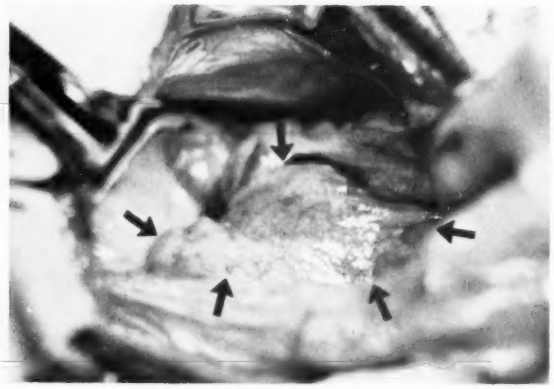

Fig. 2. The duodenal hematoma as seen on surgical exploration.

It was decided to try conservative therapy and a Levine tube with Wangensteen suction was inserted. This evacuated the stomach and the suction was continued for twenty-four hours. The patient continued to vomit in spite of a liquid diet and treatment with Sparine. On September 21, 1959, it was felt that the condition of the patient was worse and that the duodenum was completely obstructed. Surgical exploration showed considerable ecchymosis and hemorrhage in the region of the second portion of the duodenum and in the surrounding retroperitoneal tissues. The ecchymosis also involved the mesentery of the first portion of the jejunum. A large bluish brown mass surrounded the descending and third portions of the duodenum (Fig. 2). Gastrojejunostomy was performed to by-pass the obstruction and the abdomen was closed. The postoperative recovery was uneventful. The patient became asymptomatic and was dismissed on September 30, 1959.

A subsequent upper gastrointestinal series on November 11, 1959, showed the gastroenterostomy to function satisfactorily. The intramural mass which had previously obstructed the duodenum was not seen; however, in that area there was some alteration in the mucosa which was atrributed to scarring following absorption of the hematoma. There was no evidence of any obstruction at this time.

DISCUSSION

The retroperitoneal fixed position of the duodenum would appear to protect it against injury. This is not the case. Because of this fixity, the duodenum is unable to escape the impact of a blow to the abdomen which, if of sufficient force, may crush the duodenum against the vertebral column or cause avulsion of it.[10,12] It has been postulated that, when distended with fluid, the duodenum is potentially a closed loop being constricted proximally by the pyloric sphincter and narrowed distally by the overlying superior mesenteric vessels. It is, therefore, easily ruptured by even a mild direct blow if the force is applied so suddenly that the involuntary defense of the abdominal muscles has not had time to exert itself by contracting. A resulting tear in the duodenal wall may occur anteriorly or frequently posteriorly where it is entirely retroperitoneal. Traumatic rupture, while in itself is a rare condition, is much more common than submucosal hematoma. Less severe trauma may cause bleeding in the duodenal wall where it sometimes results in a hematoma large enough to produce obstruction, as in this case.

Similar submucosal hematomas have been described in the colon[3,4] and in the small bowel.[5,6] Strachan[18] in 1920 reported a case of an obstructing lesion of the duodenum visualized on an upper gastrointestinal roentgenogram which surgically was found to be due to a retroperitoneal hematoma. Oppenheimer[11] cited a case of acute obstruction of the duodenum. An autopsy later showed that the obstruction was caused by a submucosal hematoma associated with invasion from carcinoma of the pancreas. Dey[1] reported one case of duodenal obstruction of unknown cause and refers to one case which was caused by trauma. Magladry and Mathewson,[7] Snider[16] and Stirk[17] described similar surgical cases in which submucosal hematomas were found in the duodenum following trauma. It is interesting to note that most of the cases reported in the literature were in children hurt while playing strenuous games or in adults following automobile accidents. Automobile accidents appear to

be becoming more frequent and blunt trauma injuries to the abdomen are assuming even greater importance. The gastrointestinal symptoms may be overshadowed by more evident injuries of the head, chest, or extremities and, therefore, may be overlooked entirely, or the presence of an injury may be discovered so late that surgical intervention is of no avail.[9]

DIAGNOSIS

The clinical features of submucosal hematoma of the duodenum are those of the trauma initially. After the injury, the patient usually has a relatively asymptomatic period which is followed in a day or two by symptoms simulating an acute abdomen with high small bowel obstruction. Epigastric pain and persistent vomiting, such as described in the case reported, are generally present. A tender, palpable mass if sought for can usually be found in the epigastrium. Laboratory studies are probably of doubtful value except for a possible slight leukocytosis or anemia.

The roentgen features of duodenal hematoma are considered rather distinctive by Felson and Levin.[2] These consist of a smooth sharply demarcated intramural mass and a crowding together of the valvulae conniventes in the involved area, producing a "coiled spring" appearance. Similar findings have been reported in several of the other cases and by Liverud[6] in a hematoma of the jejunum. Obstruction may or may not be present. The history of trauma to the abdomen followed by gastrointestinal symptoms should make one consider the possibility of hematoma if such a lesion as described is found in the duodenum on roentgenograms. It is important to make the roentgenologic diagnosis, if possible, since surgery can be avoided, as it was in 3 of the reported cases in which complete obstruction was not present. If complete obstruction is present, surgical exploration and incision and drainage of the hematoma with or without a gastroenterostomy is probably the treatment of choice.

SUMMARY

A case of hematoma of the duodenum demonstrated roentgenologically is presented. The findings of an intramural mass obstructing the duodenum were verified at surgery. The history of trauma to the abdomen followed by more or less latent gastrointestinal symptoms and roentgenologic findings characteristic of an intramural tumor are thought to be distinctive of the lesion and allow a definitive diagnosis to be made.

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THE BIG DUODENUM*

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WHEN interference with emptying of a hollow viscus occurs, the organ dilates. So it is with the duodenum. Obstruction of the distal duodenum or jejunum results in increased duodenal peristaltic activity, hypertrophy of the wall, dilatation, and retention of contents. Benign and malignant neoplasms, atresias and stenoses, malrotations, volvuli and bands, inflammatory masses and cicatrices, adhesions, compression by vessels and aneurysms are capable of causing duodenal obstruction. All these are mechanical in nature. Not all obstructions of the duodenum are mechanical however, nor do all dilatations of the duodenum signify the presence of mechanical obstruction. To assume so, leads to mistakes in diagnosis and to error in treatment. Dilatation of the duodenum without discontinuity of the lumen has several aspects which we propose to consider in this paper.

Duodenal enlargement of this nature can be grouped into two categories. First, the duodenum may dilate secondary to a lesion distal to it. Disease of the wall of the distal duodenum or jejunum, although the lumen itself is not obstructed, may give rise to duodenal loop and bulb enlargement. Secondly, dilatation may result from interference with the contractility of the bulb and loop themselves, with the lower intestines being essentially normal.

CLINICAL FEATURES

Although pathologic enlargement of the duodenum is not precisely determined, an attempt at definition has been made. In measurements of 211 normal individuals, Meschan *et al.*[49] found that the diameter of the second portion of the duodenum was almost invariably 2 cm. The smallest diameter was 1 cm., the largest 2.5 cm. The rugae were also very constant in width, being 0.3 cm. in mid-duodenum. The duodenal bulb never exceeded 3.5 cm. at its base with a usual measurement of 3 cm. The normal bulb never exceeded 4 cm. in height from pylorus to apex with the usual value being 3 cm. The author feels that an increase in the size of the duodenum above these values should suggest a megaduodenum of significance, but it is realized that in time the limits of normality may be revised upward. The true megaduodenum must be seen repeatedly on multiple roentgenograms and at fluoroscopy. With an increase in diameter, elongation and redundancy are also observed. Folds are spaced farther apart.

The roentgen criteria for the diagnosis of megaduodenum should also include the observation of prolonged retention of barium. Peristalsis may be increased when the duodenum is still attempting to overcome a distal obstructive lesion, whether this obstruction be an organic encroachment of the lumen or improper function of a segment of bowel. If obstruction is prolonged, the proximal bowel decompensates, and peristalsis is consequently deficient. Reversed peristalsis, or the to-and-fro movement of barium in the duodenum, was once thought to indicate dysfunction. It is seen in normal people and can no longer be considered of significance. A roentgen sign of megaduodenum is a cascade effect of the barium dripping from the bulb into the dilated loop when the patient is in the erect position. To fulfill the criterion for a large, dilated duodenum of the type with which we are concerned, there must be an absence of encroachment upon the lumen by a lesion of any kind. Inflammation and ulceration in the abnormally large duodenum may be present. Some believe stasis of contents produces these changes. Others feel

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that ulceration may be primary, and localized ileus and stasis are secondary. A large amount of barium passing quickly from the stomach can temporarily distend the duodenum, thus giving the false impression of an abnormally dilated duodenum.

Symptoms associated with a huge duodenum are nonspecific and cannot be differentiated from symptoms accompanying other lesions. Epigastric discomfort, cramping pain, nausea, belching, anorexia and vomiting are described. Loss of weight would be dependent upon length of illness, melena or the accompaniment of ulceration. Recurring symptoms with intervening periods of relief are encountered. The older literature attributes migraine headaches to duodenal stasis, but this no longer appears valid. The physical examination is essentially negative.

INTESTINAL MOTOR PHYSIOLOGY

A few comments on intestinal motor physiology are in order. Two types of contractions are observed in the small intestine: (1) the rhythmic, local contractions and (2) the coordinated, peristaltic movements. The simple rhythmic bowel contractions, which have a mixing function, are largely independent of either extrinsic or intrinsic nervous control, and are thus myogenic in nature. The stronger, more complex intestinal movements have a propulsive function. Some extrinsic control is exerted over these movements by the central nervous system through the vagus nerve which carries parasympathetic system impulses, and the splanchnic nerves which carry the sympathetic impulses. Stimulation of the vagus results in increased tone and peristalsis of the small intestine. Splanchnic stimulation decreases tonus and peristalsis. Reflex inhibition of the intestinal motility experimentally is elicited by distention, heating, mechanical or electric stimulation of a jejunal loop, by peritoneal irritation, or by other stimuli. Ileus produced by experimental techniques is relieved by splanchnic division or spinal anesthesia.

Conceivably, central nervous system influences could be exerted on bowel motility through the autonomic nervous innervation. Lesions of the vagus and splanchnic nerves could also alter intestinal contractions. Propulsive contractions of the small bowel, however, occur in the absence of all extrinsic innervation. Of prime importance, then, is the intrinsic nervous system without which peristalsis does not occur and without which local enteric reflexes cannot exist. The ganglion cells and their neurones are located between circular and longitudinal smooth muscle layers, the plexus of Auerbach, and beneath the submucosa, in the plexus of Meissner.

Removal of the ganglion cells of Auerbach from the intestinal wall, or application of the local anesthetic cocaine, results in cessation of peristalsis. Nerve transmission in autonomic ganglia and muscle contraction are initiated by the release of a chemical mediator, acetylcholine, at the parasympathetic nerve ending. Rapid inactivation of acetylcholine by choline esterase insures only brief activity of the target cell as a result of the individual nerve impulse and allows the nerve to conduct the next impulse. Sympathetic system impulses are mediated by the release of adrenin at the nerve endings. In addition, the correct concentration of potassium ions is considered necessary for proper function of nerve elements.[31] Potassium ion concentration is higher within the muscle cell than in the extracellular fluid, while with sodium and chloride ions an inversion of this status exists. The difference in ion concentration is responsible for a membrane electrical potential. The membrane potential is intimately concerned with the activity of the muscle cell. When the membrane potential is low, the cells are active; at higher potentials, the muscle tension is low.[9] The actively contracting intestinal muscle cell loses potassium and gains sodium. Although the amounts involved are very small, potassium must be returned to the cell and sodium removed by certain metabolic processes if excitability is to be maintained.[9]

Intimately related with the muscle cells of the intestinal wall are the parasympathetic ganglion cells and their post-ganglionic fibers. Of the sympathetic system only the post-ganglionic fibers ramify among the muscle cells, since the ganglia are located outside the bowel wall. To view metabolic, circulatory and inflammatory lesions as causing dysfunction of nerve and not muscle, or muscle and not nerve, courts error. Inflammatory exudate and tissue and decreased vascularity reasonably act adversely on both nerve and muscle. Disease process, whatever the origin, may alter the immediate environment of nerve and muscle cell, may alter local concentration of potassium and other electrolytes, their production or destruction and transport the humoral agents. Likewise, local variation in the supply of oxygen and metabolic substrates and removal of waste products could result from disease.

Certain substances are capable of altering intestinal motility. Atropine blocks the response of smooth muscle to acetylcholine. Interference with nerve transmission is a property of eserine, which acts by inhibiting acetylcholine esterase. Hexamethonium blocks the synaptic transmission of the sensory impulses to the motor neuron.[9] Possibly drugs administered for other purposes and abnormal metabolites have a similar action, which, as yet, is poorly understood or unrecognized.

CLASSIFICATION

In this presentation megaduodenum with continuity of the intestinal lumen is classified as follows: I. Dependent upon influences mediated through the intact central and autonomic nervous systems (paralytic ileus of the duodenum): (1) pancreatitis; (2) cholecystitis, enteritis and other inflammatory entities; (3) peptic ulcer; (4) emotional states; (5) trauma. II. Dependent upon lesions in the extrinsic nervous system: (1) surgical—postvagotomy; (2) neuropathic—diabetes, vitamin deficiency, porphyria, inflammation. III. Dependent upon abnormality of nerve and muscle of the bowel wall: (1) congenital aganglionosis; (2) degenerative or toxic ganglion cell disease—scleroderma, porphyria, vitamin deficiency, electrolyte imbalance; (3) local vascular disease—lupus, purpura; (4) abdominal adhesions; (5) local muscle disease—dermatomyositis, hypoproteinemia.

I. DEPENDENT UPON CENTRAL NERVOUS SYSTEM

Duodenal participation in a generalized paralytic ileus is common. A localized paralytic ileus of the duodenum has been seen as an isolated finding in various inflammatory conditions of the abdomen, the most common of which is pancreatitis.[5,61] Duodenal atonicity and lack of mobility may be the only sign of pancreatitis[10] and may persist long after the serum amylase value returns to normal.[50]

Cholecystitis and appendicitis have also been considered as producing duodenal stasis.[62,66] Diminished or absent bowel motility is described in early regional enteritis,[47] although the duodenum is not specifically mentioned. Inhibition of the duodenum is probably mediated through the splanchnics in response to pain or irritative stimuli.

Peptic ulcer sometimes is associated with a baggy, atonic duodenal loop.[44,54] To consider this phenomenon a reflex ileus appears most logical. Possibly there is a change in the environment of ganglion cells and muscle fibers due to drainage of inflammatory products or hyperemia from an adjacent lesion and from the common retroperitoneal tissue. This possibility should likewise be considered for pancreatitis. Flabbiness of the duodenal wall and stasis of duodenal contents have been complications in the postoperative period.[57,65] Spine injury, application of a body cast, and severe trauma, in general, are known to produce duodenal ileus.[15,65]

Most controversial of all is the question of the possible production of duodenal ileus by emotional factors. Kuntz[42] speaks of flaccid intestinal obstruction arising as part of a hysterical syndrome. Friedman[25] found that emotional disturbances not only

caused the mucosal pattern of the small bowel to become disordered, but also caused hypomotility or hypermotility with variably distended and contracted loops of bowel. Marked intestinal dysfunction in anorexia nervosa patients is also described.[38]

Some persons were noted to have duodenal spasm during distressful or painful mental and physical situations, while others exhibited this finding spontaneously.[1] It could be postulated that to produce megaduodenum as a response to emotional upset, spasm of the distal duodenum would have to occur over a prolonged period. Conversely, duodenal dilatation and atonicity might be produced by emotions. A decrease in small bowel activity of as much as 24 per cent when placebos were administered has been noted.[11]

II. DEPENDENT UPON EXTRINSIC INNERVATION

Knowing that small intestinal peristalsis occurs after complete extrinsic denervation, it is difficult to envision lesions of vagus and splanchnic nerves as having much control over intestinal tone and motility. Nevertheless, duodenal dilatation is remarkably constant in the early postvagotomy period.[41] Gastric atony and dilatation of other intestinal loops are common but variable findings. Occasionally, the duodenal ileus is the prominent feature. Sympathectomy is not associated with obvious changes in duodenal tone and size. Localized dilated gut segments have been seen in diabetes and other neuropathies.[38] Whether these motility disturbances are dependent upon lesions in the extrinsic or intrinsic nervous system of the intestine, or both, is not known. The motility disturbances and dilatations seen in vitamin B deficiency[28,43] with its peripheral neuropathy could in part stem from extrinsic nerve lesions. Toxic degenerative changes in the peripheral nerves and sympathetic ganglia are reported in the ileus of porphyria.[23]

Disease of the mesentery and expansion of adjacent portions of intestine have been observed.[32] In dogs under spinal anesthesia Chesterman[12] produced a localized distensive ileus by the injection of methylated spirit between the leaves of mesentery. The distensive ileus secondary to mesenteric lesions is considered to be due to interference with extrinsic innervation. Circulatory interference more likely causes bowel spasm. In neither instance is the lumen occluded nor the bowel wall abnormal. The duodenum, having no true mesentery, is more influenced by similar lesions in the regional retroperitoneal tissue which interfere with the extrinsic nerves.

III. DEPENDENT UPON ABNORMALITY OF NERVE AND MUSCLE OF THE BOWEL WALL

Since the intrinsic nervous system is necessary for peristalsis, abnormalities of the ganglion of Auerbach's plexus are expected to be manifested by disturbed bowel motility. Probably, the most obvious example of this is the absence or the degeneration of the cells of Auerbach's plexus. Hunter[40] reported only traces of myenteric plexus in dilated duodenal and ileal loops of a stillborn infant. Megaduodenum in an adult with deficiency of ganglion cells was noted by Barnett and Wall.[4] The concept of a spastic aganglionic segment with a proximal decompensated and dilated gut is well accepted in clinical megacolon, and, to a lesser extent, in cardiospasm. Similar small intestinal lesions are rare, and lesions limited to the duodenum rarer still. If the spastic intestinal segment is very short, proximal dilatation need not occur and symptoms do not arise. The movement of the fluid contents of the upper tract is not necessarily blocked by the fading out of the wave of contraction in a short unresponsive segment. A longer unresponsive segment of small intestine more likely produces detectable dysfunction. In the colon, because of the more solid contents, a segment of altered tone and responsiveness is less well tolerated. Absence of ganglion cells and fibrosis of the wall are described in progressive systemic sclerosis (generalized scleroderma), in which lack of peristalsis and stasis in the duodenum were observed.

In 5 additional cases a striking appearance of paralysis and lack of peristalsis, particularly affecting the duodenum and jejunum, with localized widening of individual loops was recorded. Duodenal residue at twenty-four hours was present in one patient.[30] In another series, 4 of 18 cases of scleroderma showed duodenal and upper intestinal dilatation and poor motility.[35]

Intestinal obstruction due to adhesions may be more complex than mere mechanical factors. With the passage of a contraction wave, the pull of the adhesion on the bowel wall predisposes to local ischemia, in time leading to fibrosis of the wall involving the Auerbach's plexus.[33]

A wide range of ganglion cell dysfunction while the cells remain histologically normal is within the realm of reason. By the time overt changes in cell structure have occurred, the gamut of functional abnormality may have been run. Exogeneous substances, medicinals, food additives, industrial and agricultural chemicals are not all certified as innocuous, and a neurotoxic action is not impossible as a cause of megaduodenum. Many toxic agents are thought to be the precipitating factor in a patient having the basic metabolic disorder of porphyria, for example. Porphyrin injected intravenously or applied locally to the bowel interrupts normal peristalsis.[51] A paralytic ileus of acute porphyria was noted to involve the duodenum.[23] What is known to happen in patients with a defect in the pigment metabolism may also be true in others. Cruz trypanosomiasis is said to produce an interstitial inflammation which destroys the intramural plexus of the esophagus and colon.[59] Intestinal obstruction caused by a persisting contracture of a length of bowel has been described frequently in the older literature. So-called "spastic ileus" is assumed to be due to temporary influences of various types acting on the intact ganglion cells and smooth muscle.[67]

Although drugs of the atropine and banthine group act at the neural-muscle junction to cause bowel paralysis and ganglionic blocking agents, such as tetraethylammonium and hexamethonium, strongly inhibit propulsive movements of the intestine,[39] other compounds may act alone on the nerve cell or directly on the muscle cell. Benzedrene sulfate is said to relax the duodenum.[60] The action of morphine is mixed, first increasing and later decreasing duodenal tone.[2] Peristaltic waves are decreased, but it is the uncoordinated motor function which adversely affects propulsion.[13] An interesting fact is the crossing of the placental barrier by hexamethonium to cause an ileus in a newborn[36] showing that even a sheltered environment is not protection against toxic manifestation of drugs.

Disease processes involving the bowel wall are known to cause duodenal dilatation and stasis without obstruction of the lumen. Of 87 lupus erythematosus patients reported by Brown *et al.*,[7] 5 had localized ileus, 3 of these involving mainly the duodenum. Since arteritis, thrombosis, and collagen deposition are the lesions of systemic lupus, sufficient reason would seem to be present for malfunctioning of both nerve and muscle cell. The case described in this paper did not present a histologic lesion of the duodenum. Were there not diminution in ganglion cells, dysfunction on the basis of replacement of muscle by fibrous tissue affords an explanation. Dilated small bowel loops in amyloidosis have been noted, although isolated duodenal dilatation is not mentioned.[34] Muscle atrophy and fibrotic replacement were more likely responsible for the dilated duodenum in a case of dermatomyositis, since the myenteric plexus was histologically normal.[54]

Duodenal and jejunal loop dilatation in anaphylactoid purpura has been reported,[19] and a constricted third portion of the duodenum with proximal duodenal dilatation in the same disease entity has also been described.[24] In this latter case, after the acute episode had subsided, the roentgenologic appearance returned to normal.

Abnormalities of general metabolism may be reflected by disturbances of motility. Deficiencies of certain vital substances

interfere with contraction of muscle and with transmission of nerve impulse. Prerequisites for normal neural muscle function would be all the moieties which go to make up normal intracellular and extracellular environment. Many of these have not been studied, but we do have some information on the importance of proteins, electrolytes, endocrine products, carbohydrate substrate and enzymes.

Adynamic ileus in 6 patients was attributed to thiamine deficiency.[43] Experimentally, diets with vitamine B deficiency for a period of one month produced atony and dilatation of the rat's gastrointestinal tract.[28] Other workers experimenting with dogs have reported diminished bowel caliber with a hypertonic appearance.[37] Pellagra and poor nutrition were thought to be contributing factors to the enlargement of the duodenum found in 6 of 63 patients with megalosplanchnia.[22] *In vitro* experiments showed that lack of carbohydrate substrate resulted in declining muscle contractions, which were restored completely by the addition of glucose of pyruvate.[26] Pancreatectomized animals showed a delayed gastrointestinal motility, which was thought to be due to hyperglycemia since exclusion of the external pancreatic secretion caused no alteration in motility.[64] Marked degenerative changes in the ganglion cells in cases of severe dietary deficiences[45] as well as the destruction of the myenteric plexus in vitamin B deficient rats with polyneuritis[20] have been reported. Contrarily, no significant lesions were seen by Eddy and Dalldorf[18] in the plexuses of vitamin B deficient rats. Deficiencies of specific factors do not necessarily act alone. Nutritional inadequacy may predispose to secondary inflammatory changes and evidence of the latter may persist in spite of nutritional rehabilitation.[56]

In a protein deficiency state, marked delay of barium passage through the small bowel in the dog was noted.[3] Mention already has been made of the need for potassium for nerve conduction.[31] A widely dilated duodenum with moderate dilatation and stasis in the small bowel in a patient with idiopathic tetany was described by Pendergrass and Comroe.[55] Although severe thyrotoxicosis was considered to cause an enlarged duodenum,[48] in general, the bowel response to hyperthyroidism is increased intestinal tone and motility. An analysis of the case reported suggests that the large atonic duodenum might have been due to excessive prolonged morphine medication.

DISCUSSION

Until this point we have avoided the subject of organic occlusions of the duodenum, particularly those attributed to bands, acute angulation of the duodenojejunal junction, and occlusion of the mesenteric artery. Some of these diagnoses seem beyond challenge, but the question arises whether some of the patients in this group of cases were not suffering from duodenal motor dysfunctions as discussed above. Uneasiness has been expressed over the diagnosis of mesenteric artery compression[14,17,58] as well as over the unsatisfactory surgical results.[6,65] Further doubt was cast by a reported case describing the association of megajejunum with megaduodenum and the occurrence of megaduodenum and megacolon in the same patient.[4] Possibly a disservice has been done to the patient and to medical progress when, in the absence of any other finding of significance, the cause of the large duodenum has been ascribed to the relative positions of the artery and the duodenum.

Additional case reports of megaduodenum with continuity of the lumen and with exclusion of other causes of duodenal obstruction have appeared in the English literature.[8,16,17,27,29,46,52,63]

It is not the intention of this paper to minimize the significance of large duodena secondary to organic occlusion of the distal lumen, but to point out the importance of motor dysfunction of the duodenum and upper jejunum. In many cases of large duodenum, it is apparent that the

findings are not completely convincing. It is necessary to realize that the lumen of the intestine may remain open and yet intestinal contents may not efficiently pass through. Fleischner and Mandelstam[24] cite a related situation in some cases of ileostomy dysfunction.

Minimal roentgenographic changes are reluctantly accepted as of clinical importance. This is in part true because occasionally a large duodenal bulb or loop is seen in cases in which symptoms are minimal and nonspecific. The tendency then is to consider the large structure as a nonsignificant variant of normal. When roentgenologic findings become extreme, it it much easier to attribute symptoms to the physiologic and anatomic abnormality.

A revival of interest in cases of large duodenums carries with it the risk that symptoms may be unwarrantedly ascribed to this finding and treated accordingly. Duodenal ileus formerly was diagnosed often enough to suggest that the diagnosis was not always accurate. Many ills were attributed to duodenal stasis and many operations were performed to relieve patients of their supposed clinical disabilites. We do not know how many of these cases would be diagnosed identically by modern roentgen and clinical methods. Perhaps, because of improvements in nutrition and in the treatment of infections, the true incidence of duodenal ileus has decreased; however, a better knowledge of the wide limits of normality in the gastrointestinal tract and an understanding of the minor variations which can easily be confused with normal patterns can yet be obtained. We have not learned all that there is to know about the gastrointestinal tract in health and disease. A quotation from Plato here seems appropriate:

"Some things I have said of which I am not altogether confident. But we shall be better and braver and less helpless if we think we ought to inquire than we should if we indulged in the idle fancy, that there is no knowing, and no use in seeking to know, what we do not know."

REPORT OF CASES

Case 1. Huge duodenum in lupus erythematosus. This seventeen year old white female was clinically diagnosed as having lupus erythematosus for about one and one-half years before she began to complain of gastrointestinal symptoms. She had had arthralgia, hematuria and albuminuria, hypertension, a characteristic rash and protracted nosebleeds and menses. Her nephrotic syndrome was complicated by encephalopathy and convulsions. Originally, a good response to prednisone and diuril was obtained. Vomiting and complaints of anorexia and nausea were first noted in December, 1958. A month later she had poor appetite and nausea. At this time she was on a high protein diet with a low salt intake, diuril, chloroquine and dilantin, in addition to metacorten therapy.

At her last admission she complained of weakness and claimed that she vomited when she lifted her head off the bed, but despite vomiting, she was still hungry. Her nausea and vomiting were also said to be associated with thickness of tongue and difficulty in speech. She had had no recent convulsions, however. A staphylococcus septicemia, thought to be originating from an acute cellulitis of the nose, was an ominous complication.

Sixteen days after admission the patient's vomiting had become severe enough to be treated by gastric tube suction. On April 4, 1959, a gastrointestinal series showed a great duodenal dilatation and atonicity (Fig. 1). The stomach was also hypotonic. The patient's temperature rose to 101° F. and she continued a downhill course. She was given albamycin and later chloromycetin, and continued on steroids. The gamma globulin dropped to an extremely low level, as did the white blood cell count. Her hemoglobin was low but blood urea nitrogen was not elevated. She developed terminal edema and ascites, and expired a month after her hospital admission.

Autopsy. The duodenum was atonic and dilated. There was no evidence of extrinsic obstruction of the duodenum or jejunum. The mucosa was normal. The jejunum and ileum were edematous and hyperemic. No pancreatitis was found. The dilated, flabby stomach had flattened atrophic rugae. Microscopic examination showed esophagitis and necrosis of the mucosal and submucosal layers of the esophagus. The stomach submucosa showed some

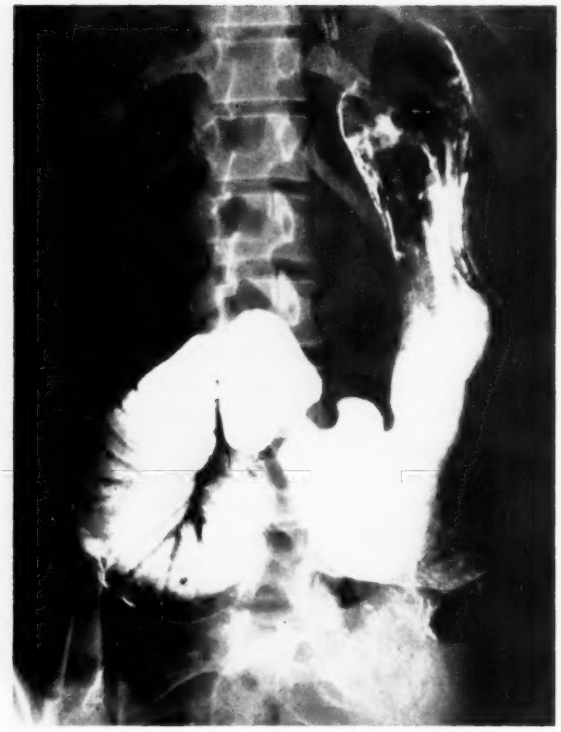

FIG. 1. Case I. Huge atonic duodenal loop and bulb in a seventeen year old female with lupus erythematosus. The stomach is hypotonic. A late roentgenogram showed very little passage of the barium beyond the duodenum. Autopsy did not reveal a gross or histologic cause for the duodenal abnormality.

edema. The duodenum was histologically normal with the ganglion cells being considered normal in number and appearance. No arterial lesions were seen in the duodenal wall. The kidney findings were consistent with lupus erythematosus. There was severe depression of both erythropoietic and granulocytic elements. Postmortem blood cultures were positive.

Comment. Insofar as present day techniques allow, no cause for the duodenal dilatation and obstruction could be demonstrated grossly or microscopically. A lesion of the autonomic nervous system extrinsic to the bowel wall would have to be considered, or possibly a central nervous system disturbance resulting in a reflex ileus involving primarily the duodenum. We have recently observed a minimally enlarged duodenum in another young woman with lupus erythematosus. She has as yet no gastrointestinal symptoms.

CASE II. Duodenal dilatation. Cause unknown. This twenty-five year old divorced female was first seen in December, 1954 with complaints of nausea and vomiting, as well as upper abdominal discomfort. At that time, the physical examination was negative and a normal gastrointestinal examination was reported, including the duodenum. The duodenum was of normal size and motility. After a short period of hospitalization, she was discharged with a diagnosis of anxiety. One year later in January, 1956, the patient again entered the hospital and complained of epigastric pain, persistent nausea and vomiting. She stated that she would vomit material that she had eaten several hours before. The epigastric discomfort was unrelieved by milk or medication. The patient admitted to heavy ingestion of alcohol. She had three children who were not living with her. Some observers thought that the patient's emotional instability had much to do with her symptoms. Roentgen examination showed a very large dilated duodenum, extending to the approximate area of the duodenojejunal junction (Fig. 2*A*). The four hour roentgenogram showed retention in the loop but normal motility of the remainder of the small bowel. The radiologist's opinion was that there was an obstruction which was incomplete. A review of the roentgenograms made one year earlier revealed the duodenum to be normal at that time (Fig. 2*B*). At examination three days later the mucosal pattern through this area could be traced. The apparent obstruction at the duodenojejunal junction was not dependent upon the erect or any other position. Cholecystography and a barium enema study were normal. All laboratory studies, including nonprotein nitrogen serum proteins, blood sugar and urinalysis were negative. Amylases were also normal. The possibility was raised by the radiologist that this duodenum, although very large, might not represent an organic obstruction of the lumen. It was decided, however, that a neoplastic lesion could not be completely excluded and surgery was therefore advised. At operation, the duodenum was found to be three times normal size. The enlargement extended down to and under the ligament of Treitz, where a narrowed area 3 cm. in length was noted. The jejunum distal to this was normal. After biopsy, this area was resected and an anastomosis was made anterior to the superior mesenteric vessels. The lumen was not ob-

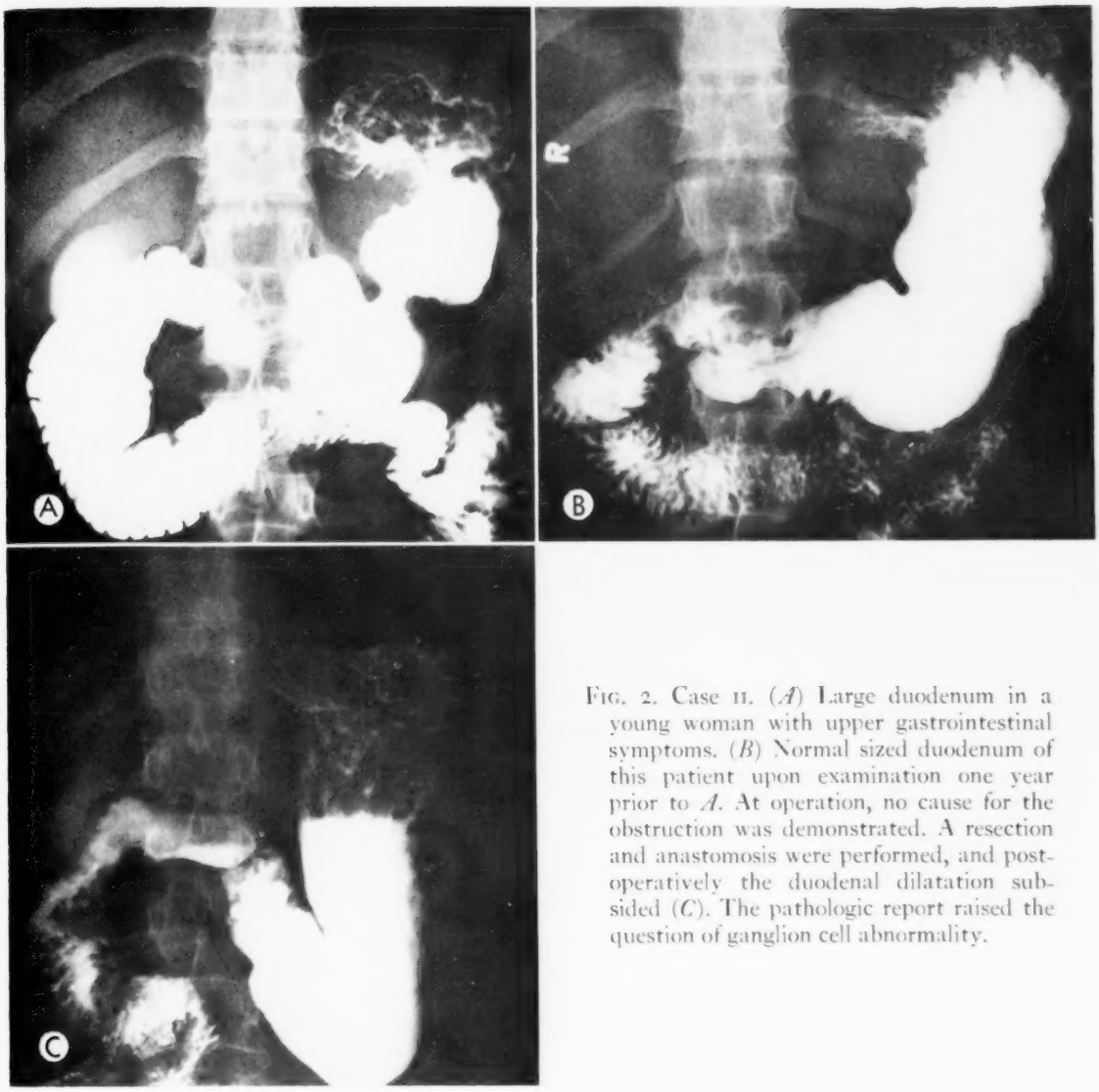

Fig. 2. Case II. (*A*) Large duodenum in a young woman with upper gastrointestinal symptoms. (*B*) Normal sized duodenum of this patient upon examination one year prior to *A*. At operation, no cause for the obstruction was demonstrated. A resection and anastomosis were performed, and postoperatively the duodenal dilatation subsided (*C*). The pathologic report raised the question of ganglion cell abnormality.

structed. Postoperatively, the patient complained of severe pain radiating to the back. Postoperative pancreatitis was suggested, but symptoms quickly improved. Ganglion cells were present in normal numbers in both Meissner's and Auerbach's plexuses. Opinion was divided as to whether the appearance of some of the cells indicated that degenerative changes were present, and if so, whether these findings could be considered primary or secondary.

One month later the patient was seen at another hospital where she complained of weight loss and continued nausea and vomiting. She stated that she occasionally vomited a small amount of red blood. Gastrointestinal roentgenograms showed that the anastomosis was open and functioning. The duodenum was considerably smaller in size. Further follow-up information has not been available.

Comment. The etiology of these upper gastrointestinal symptoms correlated with a large, dilated duodenum is unknown. In view of the normal duodenum seen on previous study, the mechanisms of duodenal dilatation may have been toxic, or may even have been emotional.

Case III. Duodenal dilatation. Question of spastic obstruction. (Courtesy of Dr. R. L. Lawton.) This thirty-eight year old white male had complaints of abdominal pain since the

Fig. 3. Case III. A greatly dilated duodenum and narrowing of the distal duodenum in a thirty-eight year old male. Operation did not conclusively demonstrate the cause of the obstruction, but there was some evidence of muscle dysfunction.

spring of 1952. He gave a history of recurrent abdominal discomfort and vomiting since the age of twelve years.

Physical examination was negative. A roentgenogram showed a greatly dilated duodenum (Fig. 3). There was constant narrowing at the junction of the third and fourth portion of the duodenum. Vigorous peristalsis was noted. An operation revealed stenosis of the duodenum just proximal to the junction of the third and fourth portion, proximal to which was marked dilatation. The surgeon's notes stated that the appearance of this area was similar to a pyloric muscle hypertrophy as noted in infants, and that there was marked thickening of the muscle fibers in this fourth portion of the duodenum. A Heineke-Mikulicz type of plastic procedure was performed on the duodenum. No tissue was obtained for histologic study. Postoperatively, there was less dilatation of the duodenum. The patient was seen at intervals and several months after the operation was reported as having no gastrointestinal difficulty of any kind. However, about a year after the operation the patient did complain of some nausea and gastric fullness with anorexia. During the next two years the symptoms gradually increased, and roentgenograms again revealed stenosis in the area previously treated. After conservative therapy, he was discharged.

In July, 1956 roentgenographic studies showed similar dilatation of the first and second portions of the duodenum with a stenotic area as seen in 1955. At operation a finger was passed through the duodenojejunal lumen and a few filmy adhesions in the lumen were broken. To the surgeon the lumen seemed adequate. A duodenojejunostomy was performed. Tissue obtained at this time showed evidence of chronic inflammation. The muscle was hypertrophied, but the ganglion cells were considered normal.

Comment. Ganglion cell disease was not substantiated by the tissue study unless the area biopsied was not the diseased segment. On the other hand, insufficient evidence of intrinsic stenosis or extrinsic bands existed.

Case IV. Large, dilated duodenal bulb in a patient with neuropathy. This sixty-one year old white female complained of postprandial fullness and bloating, and epigastric distress of several months' duration.

This patient had myxedema in 1956. After treatment with thyroid extract, she is now considered to be a low euthyroid. For the past year and a half the patient was known to have peripheral neuritis of the stocking-glove type. She had been treated elsewhere for pernicious anemia, but this diagnosis could not be definitely established here. A muscle biopsy at this hospital was normal. The anemia and neuritis were considered to be on a nutritional basis.

Physical examination was negative except for diminished pain sensation below the mid-tibial and mid-forearm areas. Proprioception and other neurologic studies were normal. The gastrointestinal roentgenogram showed a very large duodenal bulb and a normal sized loop (Fig. 4). The base of the bulb measured on different roentgenograms was 4 cm. to a maximum of 6.5 cm. The height of the bulb was 3.5 cm. to 6 cm., depending upon the projection. Both of these values are considerably higher than normal.

Fig. 4. Case iv. A very large duodenal bulb, but a normal sized loop in a sixty-one year old female with peripheral neuropathy. The base of the bulb measured 4 cm., the height 3.5 cm.

Fig. 5. Case v. Retention of barium in a dilated atonic duodenal loop is seen on the four hour roentgenogram of a fifty-seven year old female with Reynaud's phenomenon. The early roentgenograms also showed the duodenal dilatation. Diagnosis of scleroderma could not be ascertained by biopsy.

Comment. It is possible that there was a lesion of the autonomic system which could have accounted for the large duodenal bulb, since there was evidence of other peripheral neuropathy.

Case v. Dilated, atonic duodenal loop in a patient with Reynaud's phenomenon. This fifty-seven year old white female was admitted to the hospital with a typical history of Reynaud's phenomenon of both hands.

Physical examination revealed induration and a non-pitting type of swelling of both hands. Small ulcerations were present on two digits. The patient gave a history of a stomach ulcer in 1953 and an operation reportedly for carcinoma of the colon in 1945. The patient did not have any gastrointestinal symptoms at this time. A gastrointestinal roentgenogram showed a slightly deformed duodenal bulb and a duodenal loop which was dilated and atonic. The esophagus was normal. Retention of barium within the loop was noted on the late roentgenogram (Fig. 5). The biopsy of the digital skin did not confirm the diagnosis of scleroderma.

Comment. This case had some resemblance to Case iv of Hale and Schatzki.[35] Their patient had Reynaud's phenomenon three years before scleroderma was definitely diagnosed.

Case vi. Atonic duodenal loop in a patient with acute duodenal ulcer. This fifty-seven year old white male gave a history of typical acute ulcer symptoms for ten days prior to admission. Vomiting gave relief. A gastrointestinal roentgenogram showed a large duodenal ulcer and an atonic duodenal loop (Fig. 6). The measurements of the width of this duodenal loop were considerably above normal.

Case vii. This forty-five year old female gave a history of upper abdominal discomfort for at

least five years. The episodes were intermittent, and were relieved by vomiting and sometimes by straightening up and leaning backwards. Recently, pain had become more severe and vomiting much more frequent. She lost eighteen pounds in the past month.

The patient stated that a diagnosis of duodenal ulcer had been made two years previously. A cholecystectomy was performed eight years before for similar upper abdominal symptoms when it was known that gallstones were present. An ulcer history was present in her father and siblings.

An enlarged, elongated duodenal loop was revealed at fluoroscopy and on the initial roentgenograms. The mucosal pattern of the duodenojejunal junction was uninterrupted. Roentgenograms thirty minutes later revealed a normal loop, but a roentgenogram at four hours showed retention of barium in the loop. The duodenal bulb was small and deformed, but no crater was seen.

Operation demonstrated a duodenal ulcer, just distal to the pylorus, penetrating into the under surface of the liver. The duodenojejunal junction was carefully examined. No obstruction was found. A subtotal gastric resection was performed.

FIG. 6. Case VI. A dilated duodenal loop in a fifty-seven year old male, during an acute duodenal ulcer episode.

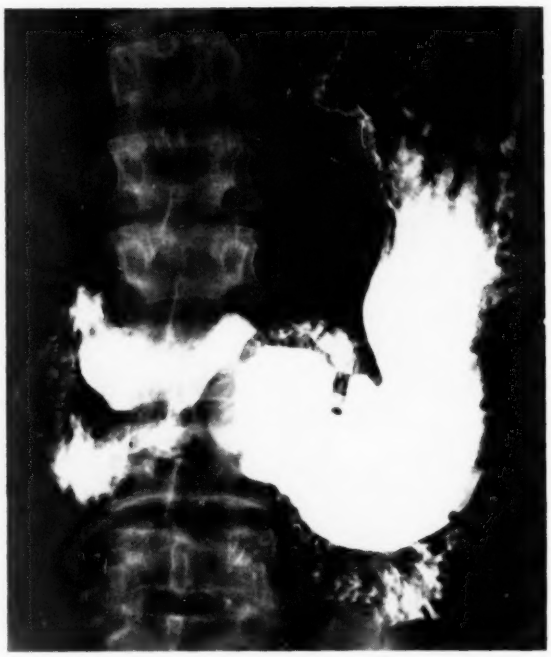

FIG. 7. Case VIII. Large duodenal bulb in a patient with vague, nonspecific symptoms. Whether this bulb is a pathologic finding or an extreme variant of normal is not known.

Comment. The findings, both clinical and roentgenologic, suggested an intermittent obstruction at the duodenojejunal region, possibly due to a spastic contracture of the bowel. The duodenal bulb in this patient could not dilate because of its scarred state. Also interesting was the patient's claim of relief of symptoms by leaning backwards. Were the symptoms ascribed to occlusion of the duodenum by superior mesenteric artery, the erect position with exaggerated lordosis should have made symptoms worse.

CASE VIII. Large duodenal bulb, cause unknown. This thirty-six year old white male farmer complained mostly of left lower chest pains which he had experienced intermittently for three years. He considered himself to have always had a "nervous stomach" and had bouts of intestinal symptoms since childhood. Symptoms were relieved by Tums and 7-Up, causing eructation. His general health was good. There was no relief from milk and bland foods. The patient experienced occasional fullness in his left side at night, which was relieved

by turning on his right side. There were no other significant findings in the history. Physical and laboratory work-ups were negative. The gastrointestinal roentgenogram showed a large duodenal bulb which was well beyond the limits of normal as stated (Fig. 7).

Comment. This is the type of case in which it is difficult to decide whether a large duodenal bulb is still within normal limits, or whether there is some esoteric reason for the large bulb. At present, it would be hard to say that his symptoms were due to the large duodenal bulb.

SUMMARY

The subject of duodenal enlargement without discontinuity of the lumen has been reviewed. Megaduodenum of this nature is caused by either paralytic motor dysfunctions of the duodenum itself or duodenum enlargement secondary to distal spastic obstructions. Illustrative cases are presented.

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THE ROENTGENOLOGIC DIAGNOSIS OF GASTROCOLIC AND GASTROJEJUNOCOLIC FISTULAS

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GASTROJEJUNOCOLIC fistula represents an integral part of the problem of peptic ulcer and its management. It is one of the most severe sequelae of the surgical treatment for this disease. The first really comprehensive report of this condition was written by Jean Verbrugge[14] in 1925 at the Mayo Clinic. Subsequently, large series of cases have been reported by Atwater, Butt, and Priestley[2] (Mayo Clinic) in 1943; Lowdon[8] (University of Edinburgh) in 1953; Localio, Stone, and Hinton[7] (New York University) in 1953; and Marshall and Knud-Hansen[11] (Lahey Clinic) in 1957.

This paper presents a recent series of cases of gastrojejunocolic and gastrocolic fistulas seen at the Mayo Clinic, with analysis of the associated roentgenologic findings.

GENERAL CONSIDERATIONS

The great majority of gastrojejunocolic fistulas result from perforation of a jejunal or gastrojejunal ulcer located at or near the site of a gastroenterostomy opening established previously in treatment of a duodenal ulcer. It is estimated that "marginal" ulcers form in about 15 per cent[11] of all patients who undergo gastroenterostomy, and that gastrojejunocolic fistula eventuates in about 14 per cent[2] of that number. The incidence is lower in patients whose duodenal ulcer has been treated by partial gastric resection rather than gastroenterostomy,[2,8,11,13] the major factor of difference being the better relief of ulcer diathesis by partial gastrectomy. Infrequently, large malignant lesions of the stomach or colon may cause formation of gastrojejunocolic fistula and in 1 reported case the fistula occurred with islet cell tumor of the pancreas.[10]

Gastrocolic fistulas result primarily from perforation through carcinomas of the stomach or colon—usually in the larger, more advanced neoplasms. Some authors[11] have stated that carcinoma of the colon is a more frequent cause than carcinoma of the stomach. Other rare causes for such fistulas have been reported, including benign gastric ulcer,[5] chronic ulcerative colitis,[1] and carcinoid tumor of the colon.[9] The incidence of gastrocolic fistulas has been reduced in recent years by early diagnosis and treatment of intra-abdominal malignant growths.

Gastrojejunocolic fistulas occur almost exclusively in men: of more than 400 cases reported up until 1958, only 6 occurred in women.[3] This marked sex difference is not found with gastrocolic fistulas secondary to malignant change. Fistulas of that kind may occur at almost any age, but the majority are found in patients forty to fifty years old. The time between the original surgical treatment and the formation of a gastrojejunocolic fistula may range from a few months to more than twenty years, the average being about five years.[2,4]

In a patient who has had a gastroenterostomy or who has an abdominal malignant lesion, the classic symptoms of diarrhea, loss of weight, and fecal eructation or vomiting strongly suggest the presence of a fistula between the stomach and the colon. Roentgenologic techniques, however, provide the most accurate means for diagnosis. An examination with a barium enema can demonstrate the fistula in almost 100 per

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cent of instances, but a barium meal study frequently fails to provide a diagnosis.[8,11] Although no anatomic explanation for such a distinction has been found, most of these fistulas apparently permit the passage of material, including barium, only in a retrograde fashion from the colon into the small bowel and stomach. During barium enema examination, the roentgenologist notes filling of the stomach and jejunum with barium from some point in the left transverse colon. Often the exact site of the fistula is difficult to determine roentgenologically, and usually one cannot distinguish between the gastrocolic and gastrojejunocolic types. Marshall and Knud-Hansen[11] found that a correct preoperative diagnosis of fistula was made in 89.7 per cent of instances of the gastrojejunocolic type, and in 55 per cent of gastrocolic type. This difference probably is due to a higher index of suspicion—and, therefore, more frequent use of barium enema examination—in the cases of symptomatic patients known to have had gastroenterostomy than in cases of abdominal malignant growths.

MATERIAL FOR STUDY

During the period 1947 to 1958 inclusive, 66 gastrojejunocolic or gastrocolic fistulas occurring in 65 patients were seen at the Mayo Clinic. Roentgenograms were available for study in 60 instances (59 patients) which comprise the series for the present study. The presence of a fistula was established surgically in 58 instances, the diagnosis being made on clinical and roentgenologic grounds in the remaining 2. All the fistulas linked the stomach and the left portion of the transverse colon, except one that connected the stomach and the transverse colon near the hepatic flexure. Eighteen of the fistulas resulted from perforation in intra-abdominal malignant lesions, and 42 occurred in patients with diseases not neoplastic. The roentgenologic aspects of these 2 groups of cases will be considered separately.

FINDINGS AND COMMENT

Group With Malignant Disease. In the group of 18 patients with malignant disease were 14 men and 4 women of ages ranging from forty-two to eighty-one years (Table 1). Twelve patients had carcinoma of the transverse colon and 6 had carcinoma of the stomach. In 12 cases (67 per cent) a diagnosis of gastrocolic or gastrojejunocolic fistula was made roentgenologically (Table II and Fig. 1, *a* and *b*), but no fistula was dis-

TABLE I

CERTAIN CLINICAL FEATURES IN 60 CASES OF GASTROCOLIC AND GASTROJEJUNOCOLIC FISTULA

Clinical Features	Types of Associated Disease			
	Malignant		Nonmalignant	
Cases	18		42*	
Sex Distribution				
Male	14		38	
Female	4		3	
Originating Lesion	Carcinoma of colon	12	Marginal ulcer	41
	Carcinoma of stomach	6	Chronic ulcerative colitis	1
Previous operation	None		Partial gastrectomy	10
			Gastroenterostomy	31
			None†	1

* Two fistulas occurred in one patient.
† Chronic ulcerative colitis.

TABLE II

SUCCESS OF ROENTGENOLOGIC DIAGNOSIS IN GASTROCOLIC AND GASTROJEJUNOCOLIC FISTULA

Roentgenologic Method	Type of Associated Disease		
	Malignant	Nonmalignant	Both Types
Barium Enema	11/13 (85%)	30/30 (100%)	41/43 (95%)
Barium Meal	3/9 (33%)	9/35 (26%)	12/44 (27%)
Total	12/18 (67%)	34/42 (81%)	46/60 (77%)

coverable by that means in 6 (33 per cent). Of the 6 patients in whom no fistula was seen, 4 were given barium by mouth only, with failure to fill the fistula at examination. The remaining 2 had barium enema examinations: demonstrated in one was complete obstruction to the flow of barium in the transverse colon; and in the other, partial obstruction in the transverse colon with barium not entering the fistula. Complete or partial obstruction in the transverse colon with a demonstrable fistula was noted in 4 other patients in this group of 18, although colonic obstruction was not found in the nonmalignant group. The combination of the two findings, namely, fistula and obstruction, is a strong suggestion of cancer as the underlying cause, as Rafal and Shaw[12] have pointed out.

A correct diagnosis was made by means of barium enema in 11 (85 per cent) of 13 patients thus examined, and a fistula was demonstrated by barium meal examination in only 3 (33 per cent) of 9 patients studied. The exact site of the fistula frequently was difficult to demonstrate roentgenologically, but it was thought to be between the stomach and left transverse colon in 11 instances and near the hepatic flexure in 1.

Group With Nonmalignant Disease. The second group was comprised of 42 instances of fistula occurring in 41 patients with non-

FIG. 1. (*a*) A small irregular fistulous tract on the greater curvature is evidenced. Barium could not be forced into the colon. (*b*) Indication is given of free communication from the colon to the stomach via a gastrojejunocolic fistula secondary to recurrent carcinoma of the colon.

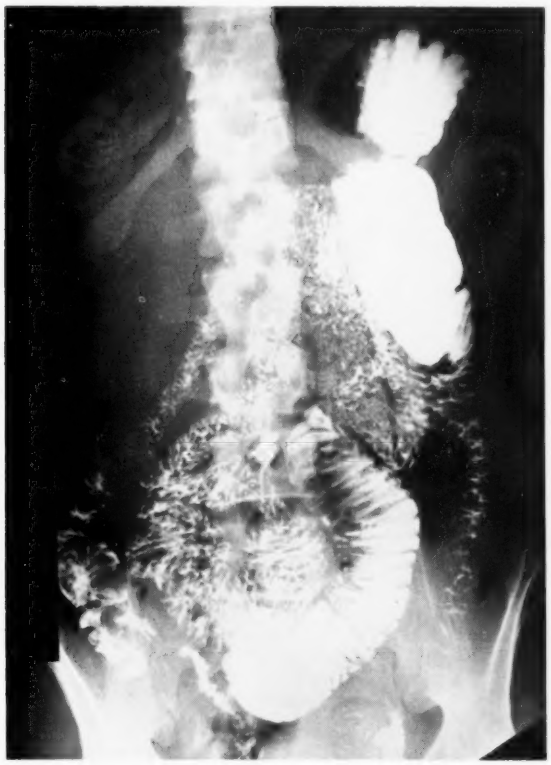

Fig. 2. Roentgenogram of the colon shows a gastrojejunocolic fistula occurring in a patient who previously had had a partial gastrectomy.

malignant disease, of whom 38 were men and 3 women, with ages ranging between twenty-one and seventy-six years (Table I). Forty-one of the fistulas had been formed by perforation of a jejunal or gastrojejunal ulcer in patients previously operated on for duodenal ulcers. The previous operations were gastroenterostomy in 31 instances and partial gastrectomy in 10 (Fig. 2). One fistula occurred in a patient with chronic ulcerative colitis.

A correct diagnosis was made roentgenologically in 34 (81 per cent) of the 42 cases (Table II), but no fistula was seen roentgenologically in the other 8. Barium enema examination was performed in 30 cases with demonstration of the fistula in every instance. Among 35 instances of examination with barium by mouth, fistula was reported initially in only 4 (Fig. 3). Review of the roentgenograms, however, revealed evidence of a fistula in an additional 5 cases, bringing the total to 9 (26 per cent). No fistula could be demonstrated in the remaining 26 patients (74 per cent) by this means. All the fistulas occurred between the stomach and the left portion of the transverse colon, although the exact site often could not be determined roentgenologically.

Additional roentgenologic features noted during barium meal examinations included enlarged gastric rugae in 21 (60 per cent) of the 35 patients thus studied. This finding was not present in any of the patients whose fistulas were secondary to malignant disease. Dilatation of one or more jejunal loops was noted in 13 of the 35 patients mentioned, a diagnosis of jejunitis was made in 9, and a diagnosis of deficiency pattern in 1. Gastroenterostomy function was considered abnormal in 12 (34 per cent) and normal in 23 (66 per cent). The examiner's appraisal of gastroenterostomy function had no direct relation with the presence or absence of dilatation in jejunal loops, jejunitis, or marginal ulcer. A jejunal or gastrojejunal ulcer was found surgically in 25 of the 35 patients examined with

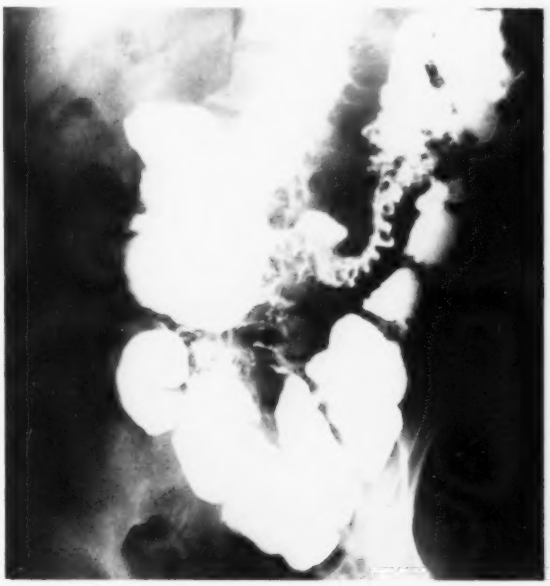

Fig. 3. A large anastomotic ulcer is near the site of a previous gastroenterostomy. Also, a gastrojejunocolic fistula with partial filling of the colon is discernible.

barium given by mouth, but was seen initially by roentgenologic means in only 9 instances. Review of the roentgenograms revealed 5 additional cases in which marginal ulcer was identifiable, bringing the total to 14 (56 per cent of the 25). No ulcer was demonstrable roentgenologically in the remaining 11 cases (44 per cent). In 5 instances marginal ulcer was diagnosed roentgenologically but not found at operation.

Errors in Diagnosis. In a total of 7 instances during the period 1947 to 1958, gastrocolic or gastrojejunocolic fistula was diagnosed roentgenologically but was not found surgically. In 3 of the 7 patients a direct connection between the stomach and lower ileum inadvertently had been established surgically—in effect, gastroileostomy. According to Gross and Waugh,[6] the signs and symptoms encountered in such circumstances may be entirely similar to those accompanying gastrojejunocolic fistula. During examination of a patient with a gastroileal stoma, barium enters the colon rapidly after ingestion or fills the stomach during barium enema examination. Careful fluoroscopic observation of the advancing barium column during barium enema examination, however, reveals that a short segment of ileum always fills before the barium enters the stomach. The mechanism may be even more readily apparent after barium is given by mouth, when the colon will be seen to fill from a short segment of ileum rather than directly from the stomach, as in gastrojejunocolic fistula.

A fourth patient among the 7 had had ileocolostomy performed subsequent to removal of all but a few feet of small intestine. Roentgenologically this situation resembled that of the patients who had had gastroileostomy.

Two patients had gastric obstruction and retained barium in the stomach from a previous examination. Detection of this retained barium during the subsequent barium enema examination led to a mistaken belief that the stomach had filled from below via a gastrocolic or gastrojejunocolic fistula. Rapid fluoroscopic evaluation of the abdomen just prior to barium enema examination would obviate such diagnostic errors.

The last patient in this group was found at operation to have a fistula from the stomach and another from the colon, both connecting with an abscess cavity in the left upper quadrant. During barium enema examination, the stomach was thought to fill directly from some point in the left portion of the transverse colon. Beyond doubt, the actuality must have been retrograde flow of barium from the colon through the abscess cavity into the stomach rather than via a direct communication between the stomach and colon.

SUMMARY

The roentgenologic findings in 60 cases of gastrocolic and gastrojejunocolic fistula seen at the Mayo Clinic, 1947 through 1958, are presented. Eighteen cases were secondary to intra-abdominal malignant disease, 41 resulted from perforation of a jejunal or gastrojejunal ulcer subsequent to operation for duodenal ulcer, and 1 case represented a complication of chronic ulcerative colitis. Barium enema examination proved the most accurate roentgenologic means of diagnosis. Seven additional cases are described in which an incorrect diagnosis of fistula was made roentgenologically when none existed.

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SYMPTOMATIC MECKEL'S DIVERTICULUM OF UNUSUALLY LARGE SIZE*

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MECKEL'S diverticula are said to occur in 1 to 2 per cent of autopsies, but are only rarely described in the clinical literature. The complications resulting from the presence of a Meckel's diverticulum are well known and the diagnosis is often entertained in obscure abdominal illnesses, especially those with bloody stools. The preoperative recognition of a suspected Meckel's diverticulum by clinical or roentgenologic methods is, however, notoriously uncertain. In the following case an unusually large Meckel's diverticulum was demonstrated roentgenographically. To the best of our knowledge, it is the largest such structure yet recorded.

REPORT OF A CASE

A sixty-eight year old obese white female was admitted on February 14, 1959 to the medical service of Dr. Frank Gregg, complaining of diffuse abdominal cramps of four years' duration. These occurred almost daily and were associated with bloating and distention, beginning several hours after eating. Vomiting relieved the distention. At times, the distress radiated to the right upper quadrant, then to the right flank and to the right scapula. The attacks were aggravated by raw foods, cabbage and chocolate. An upper gastrointestinal tract study performed about four months prior to admission was said to have shown pylorospasm. Antacids were then prescribed but produced only questionable relief. There was no history of bleeding from any body orifice.

The past medical history included a uterine suspension in 1932, an operation for "back pain" in 1934, an abdominal hysterectomy in 1938, an operation upon the gallbladder for cholelithiasis (possibly cholecystectomy) in 1940, and a probable myocardial infarction in 1954.

The entire physical examination was essentially normal. Three well-healed abdominal scars were present and abdominal peristaltic activity was estimated to be within normal limits.

An electrocardiogram was interpreted as showing a probable old posterior infarction with moderate coronary arterial disease and damage to the right ventricular myocardium. Free gastric acid was present in the stomach. Urinalysis and complete blood cell counts as well as routine chemical serologic tests were negative except for a mild hypochromic anemia and slight elevation of serum lipase.

The diagnostic impressions on admission listed possible pancreatitis, biliary tract disease, duodenal ulcer, or gastrointestinal malignancy, and irritable bowel syndrome.

Chest roentgenograms were essentially normal. The biliary tract was investigated in succession by oral cholecystography, intravenous cholangiography and combined oral and intravenous cholangiography, resulting in nonvisualization of the gallbladder and demonstration of a normal common duct without filling defects or obstruction. An upper gastrointestinal tract study was entirely negative with respect to esophagus, stomach and duodenum, but on several roentgenograms (Fig. 1) a large gas-filled left upper quadrant pouch was seen which eventually filled with barium following opacification of the jejunum and a portion of ileum. The first barium enema study (Fig. 2) showed a large collection of barium in the left upper quadrant pouch which was not tender, contained few mucosal markings, filled from a loop of ileum and produced no significant displacement of the descending colon. A questionable sigmoid polyp was also noted and re-examination was suggested. The second barium enema examination (Fig. 3) again demonstrated the large pouch; the colon appeared normal without filling defects. The sigmoid was somewhat irritable. Review of all the available examinations revealed a large left abdominal pouch filled with barium and air in

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various positions which extended from the inferior surface of the left diaphragm along the lateral peritoneal surface to the floor of the pelvis, and filled by way of a single communication with the mid-portion of the ileum. It was characterized by smooth, flat surfaces with minimal mucosal markings. It was thought to represent either a very large enterogenous cyst or segmental reduplication of the ileum or a giant diverticulum of the mid-ileum. A diverticulum resulting from lymphosarcoma was considered as highly improbable.

Surgical exploration and removal of the lesion were recommended and the patient was re-admitted to the surgical service of Drs. John McAleese and Ralph C. Wilde. Operation on March 19, 1959 revealed a tremendously large diverticulum arising from the antimesenteric border of the terminal ileum, approximately 25 cm. proximal to the ileocecal valve, and possessing its own mesentery. Its blood supply consisted of prominent vessels derived from the ileal mesentery and crossing over the ileal surface at the site of attachment of the diverticulum. The diverticulum proper measured 10 cm. in diameter and 18 cm. in length (Fig. 4). It was thin-walled and contained fluid. The abdominal cavity was free of any adhesions and the diverticulum could be maneuvered into any portion of the peritoneal cavity. Because the ostium of the diverticulum was large, the corresponding segment of ileum was resected and intestinal continuity restored by end-to-end anastomosis. After a satisfactory immediate postoperative course, the patient developed a mechanical low small bowel obstruction, decompressed by means of a Cantor tube and subsequently a pelvic abscess which required drainage per rectum. She then recovered uneventfully and was discharged on April 16, 1959 in good condition.

Fig. 2. Barium enema study shows filling of part of Meckel's diverticulum from ileum (arrows indicate ostium).

Fig. 1. Bucky posteroanterior roentgenogram following upper gastrointestinal examination shows beginning opacification of Meckel's diverticulum from ileum (arrows indicate ostium).

On her most recent return visit, two months following discharge, she has remained completely free of her preoperative symptoms.

The pathologic report describes a large blind pouch of small intestine with a thin wall and only a few thin mucosal folds. The thickness of

FIG. 3. Barium enema postevacuation roentgenogram in prone position shows entire diverticulum outlined by retained barium with superimposition of a portion of the descending colon.

the wall consisted of all four layers normally found in the intestine. The mucosa was of the small intestinal type with eosinophilic staining of the cytoplasm of the lining cells. No ectopic tissues were seen. The pathologic diagnosis was diverticulum of the small intestine (Meckel's).

DISCUSSION

Meckel's diverticulum is an embryologic remnant of the vitelline duct and when present, usually consists of a small pouch arising from the antimesenteric border of the mid-ileum. For a complete review of the subject, the reader is referred to Berne's[1] excellent and comprehensive review. The greatest length of diverticulum recorded by Berne is 13 cm. Another unusually large Meckel's diverticulum was described by Marshak and Friedman[2] and measured 9×10 cm. at operation. This lesion had been diagnosed roentgenologically; at operation it was surrounded by numerous inflammatory adhesions and had produced a tremendous degree of small intestinal obstruction.

Differentiation of Meckel's diverticulum from enterogenous cyst or duplication of the small intestine in this area is not possible by roentgenologic means and is rather academic in any event. Both of these structures may give rise to identical appearances and symptoms. The specific differentiation can be accomplished by the surgeon at the time of the operation since

FIG. 4. Photograph of the fresh surgical specimen.

duplication of the intestinal tract of necessity arises on the mesenteric border of the normal bowel, whereas the Meckel's diverticulum characteristically arises from the antimesenteric border. The corresponding blood supply and mesenteric attachment of the pouch follow a distribution resulting from the congenital derivation of these structures. Histologically, both Meckel's diverticula and reduplications contain all four component layers of intestinal tract, but acquired diverticula do not.

The case described above is unusual not only from the standpoint of its very large size but also because of its remarkable symptomatology, a logical result of the presence of a large distensible pouch. Furthermore, one should note the total absence of adhesions or other evidences of the usual complications associated with symptomatic Meckel's diverticulum. It may be assumed that perforation, torsion, or ulceration of such a structure are apt to occur at any time, as well as mechanical small bowel obstruction. Hence, prophylactic resection appears advisable once the lesion is known to be present. In our case, however, the symptomatology was sufficiently severe to make removal of the diverticulum mandatory.

From the roentgenologic diagnostic standpoint, suffice it to emphasize again that small Meckel's diverticula can rarely be demonstrated in a small bowel study. Such structures may be identified if barium is retained within them and their mucosal markings can be distinguished from the surrounding loops of small intestine. Obviously, the larger the pouch, the more easily will it be recognized.

SUMMARY

A case of unusually large Meckel's diverticulum diagnosed preoperatively and removed surgically is reported because its size was much greater than that in any previously described cases and because of the specific symptoms which it produced. Roentgenologic differentiation of Meckel's diverticula from enterogenous cysts or duplications of the intestinal tract is impossible. The differentiation as discussed in this report is readily made during surgical operation, by virtue of the characteristic differences in embryologic derivation of these lesions.

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Permission by Dr. Frank Gregg to report this case is hereby gratefully acknowledged.

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FINDINGS ON PLAIN ROENTGENOGRAMS OF THE ABDOMEN ASSOCIATED WITH MESENTERIC VASCULAR OCCLUSION WITH A POSSIBLE NEW SIGN OF MESENTERIC VENOUS THROMBOSIS*

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MESENTERIC vascular occlusion has been recognized as a clinical entity for more than a century. Since the first case report by Teidman in 1843, as quoted by Mersheimer *et al.*,[5] over 200 articles have appeared in the medical literature describing various facets of this entity. However, there is still considerable difficulty in diagnosing this disease. The increasing use of routine plain roentgenograms in the examination of the acute abdomen has resulted in the recognition of several signs which are highly suggestive, although not diagnostic, of mesenteric vascular occlusion. Frimann-Dahl[2] has called attention to the "pseudotumor" sign due to the water-density mass seen on plain roentgenograms of the abdomen, apparently caused by the marked amount of fluid in the infarcted bowel lumen. Others have called attention to the roentgenologic manifestations of ileus, the extent of which corresponds to the distribution of the occluded mesenteric vessel. We wish to call attention to an additional roentgenologic sign which was recently observed in two proven cases of mesenteric venous thrombosis at The Ohio State University Hospital.

ETIOLOGY AND PATHOLOGY

The term "mesenteric vascular occlusion" includes both thrombosis of the mesenteric vein and embolism or thrombosis of the mesenteric arteries. Venous thrombosis is reported as being more common than arterial occlusion,[4] although some have reported the reverse. Arterial thrombosis is more common than arterial embolism and occlusion of the superior mesenteric artery occurs far more frequently than occlusion of the inferior mesenteric artery.

The etiologic classification of mesenteric venous thrombosis includes both idiopathic and secondary causes, the majority of cases belonging to the latter group. The idiopathic factor is postulated as being due to an inherent and unexplained defect in the coagulating mechanism. There are several precipitating factors of secondary mesenteric venous thrombosis. For example, intestinal obstruction may increase the intraluminal pressure to a level sufficient to obstruct or produce considerable stasis in the flow of blood through the venous network in the wall of the obstructed loops. Intra-abdominal infections, such as those seen following a perforated appendix, pelvic abscess, etc., are thought to play an important role in causing mesenteric venous thrombosis. Portal cirrhosis may cause a marked increase in the portal venous pressure with resultant venous stasis, thus favoring the development of venous thrombosis. Blood dyscrasias are also associated with abnormal clotting tendencies, thought to be a factor in some cases of mesenteric arterial and venous thromboses.

Embolic occlusion of the mesenteric arteries may occur from emboli originating from the left atrium in patients with atrial fibrillation, or those originating in the left ventricle following a myocardial infarction. Atheromatous emboli may become dislodged from the aorta and enter the superior or inferior mesenteric artery. Mesenteric arterial thrombosis most frequently

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occurs within vessels that are markedly atherosclerotic, particularly in patients with associated hypertensive cardiovascular disease. An aneurysm of the abdominal aorta closely associated with the orifice of the superior mesenteric artery, or an aneurysm of the superior mesenteric artery itself, may precipitate a mesenteric arterial thrombosis. In both the venous and arterial occlusions gross examination of a segment of involved bowel will show infarction of the bowel wall. However, in general, the bowel wall is more thickened in the mesenteric venous occlusion, and there is more blood and fluid within the lumen. In the case of mesenteric arterial occlusion, necrosis of the bowel wall and mucosal ulceration are common. Both arterial and venous types of occlusion are associated with blood and fluid within the bowel lumen, although there tends to be more blood in cases of mesenteric venous occlusion. This fact probably accounts for the marked degree of hypovolemia and shock seen clinically. Microscopic examination of the infarcted bowel in both the venous and arterial occlusions shows marked degeneration and ulceration of the mucosa, with an extensive hemorrhagic reaction of the serous and muscular layers, where the blood vessels are dilated and engorged with red blood cells.

Occlusion of the superior mesenteric artery causes an abrupt and catastrophic onset progressing rapidly to death. In contrast, occlusions of the mesenteric vein usually begin insidiously and usually progress over a period of twenty-four hours, although some cases may progress slowly for a week or longer. A dominant feature, particularly in the arterial occlusions, is constant severe abdominal pain sometimes causing an intense lumbar backache which is not relieved by narcotics. This pain, which may occur without associated clinical signs of peritoneal irritation, is believed to be true visceral pain secondary to vascular anoxemia. Diarrhea or constipation may occur, although diarrhea usually predominates. Melena may be present, and is more frequent in the mesenteric venous occlusion. Moderate abdominal distention is often evident, and the degree of tympany depends upon the quantity of air within the small bowel loops. Tympany may be absent and a poorly-defined boggy mass (the "pseudo-tumor") may occasionally be palpated if the loops of involved intestine contain a large amount of fluid.

ROENTGENOLOGIC FINDINGS

In 1944 Rendich and Harrington[6] described their roentgenologic observations in 3 proved cases of mesenteric venous thrombosis. In 1947 Harrington[3] added 4 similar cases. In their opinion the most striking roentgen finding was localized distention of the intestines simulating a mechanical obstruction but having a distribution which corresponded to that of the superior mesenteric artery with an abrupt demarcation of the distended intestine near the splenic flexure of the colon. In one case a barium enema examination showed no obstruction distal to the distended intestine. They concluded that the diagnosis of mesenteric venous thrombosis should be considered when plain roentgenograms of the abdomen demonstrate a dilated small bowel and colon to the region of the splenic flexure and a subsequent barium enema study fails to show any evidence of mechanical obstruction.

Frimann-Dahl[2] described his observations in the same disease and stated that in his experience only a few cases confirmed the observations made by Harrington. He noted the gaseous distention of the colon to be demarcated more distinctly at the hepatic flexure. He pointed out that mesenteric vascular occlusion should not be confused with mechanical obstruction of the small intestine, for in such conditions there is practically no gas present in the colon. He believed that examination after oral barium sometimes gave more reliable criteria for the diagnosis. He noted that when the mesenteric vascular occlusion affected the small bowel mainly, a remarkable retention of the contrast material was seen in the in-

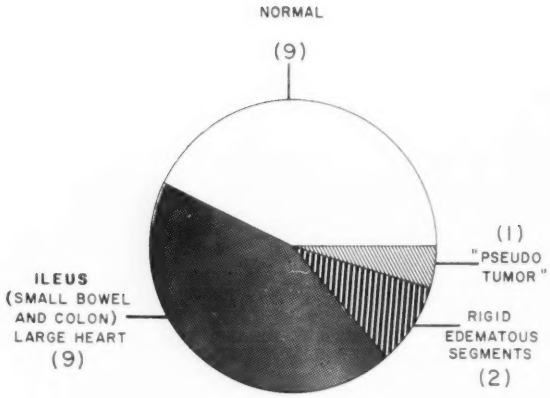

FIG. 1. Findings in 21 proved cases of mesenteric vascular occlusion. The numbers within the parentheses indicate the number of patients showing the described roentgen findings. Note that 12 of the 21 patients had definitely abnormal findings including the "pseudo-tumor" and "rigid loop" signs.

volved loops. Utilizing this method of oral barium administration, he also pointed out that the intestinal loops are not dilated as in pure mechanical obstruction, and that the mucosal pattern showed signs of thickening and rigidity due to edema. The folds are relatively thick and close to each other with irregularly serrated contours. A supine roentgenogram twenty-four hours later sometimes shows retention of barium in the involved loops. Inflammatory disease may produce a similar mucosal pattern, but usually involves the upper loops of jejunum or terminal ileum, most often leaving the remainder of the bowel unaffected. There may be retention of barium in such cases, but it will reach the cecum in the course of eight to ten hours, in contrast to the marked delay in cases of mesenteric vascular occlusions.

Among the 49 cases of mesenteric vascular occlusion seen at The Ohio State University Hospital since 1950, we were able to review the roentgenograms of 21 patients (Fig. 1). This series is admittedly small, but does show that, in spite of relatively incomplete roentgenologic procedures in many instances, we were able to detect definitely abnormal findings on plain roentgenograms of more than one-half of the patients whose films were available. Many of these abnormal roentgenologic findings were nondiagnostic of mesenteric vascular occlusions, although the diagnosis was suspected in a few patients on the basis of stasis (ileus) of the small bowel and right colon, corresponding to the distribution of the superior mesenteric artery and vein. Furthermore, a typical strikingly large "pseudo-tumor" sign was seen in one patient, and this was highly suggestive of the diagnosis, inasmuch as it occurred in a female of average size who undoubtedly would have been aware of such a large mass if it were due to a previously existing neoplasm. However, this patient had not been aware of any previously existing intra-abdominal mass of the size of the "pseudo-tumor." Other positive findings in our cases consisted of nonspecific types of ileus localized to various areas of the small intestine and right colon.

THE "RIGID LOOP"—A POSSIBLE SIGN OF MESENTERIC VENOUS OCCLUSIONS

Of great additional interest to us was the observation in 2 patients of curvilinear collections of gas in a single loop or two adjacent narrow-lumen loops of small bowel (Fig. 2, *A* and *B*; and 6, *A* and *B*). The configuration and location of these collections of gas did not change in one patient from one day to the next (Fig. 2, *A* and *B*; and 3, *A* and *B*). In normally distensible hollow viscera, or in the hollow viscera involved in paralytic ileus or proximal to mechanical obstructions, the air and fluid contents of the involved loops obey the laws of gravity. The distensibility of the still pliable walls of the affected loops is manifested by the fact that fluid will flow into and distend the most redundant portions of the loops, whereas air rises and distends the uppermost portions of the loops (Fig. 7, *A* and *B*). The segments of the loops between the most redundant fluid-filled portions and the uppermost air-filled portions probably become stretched and of smaller caliber to a degree proportionate to the increase in size

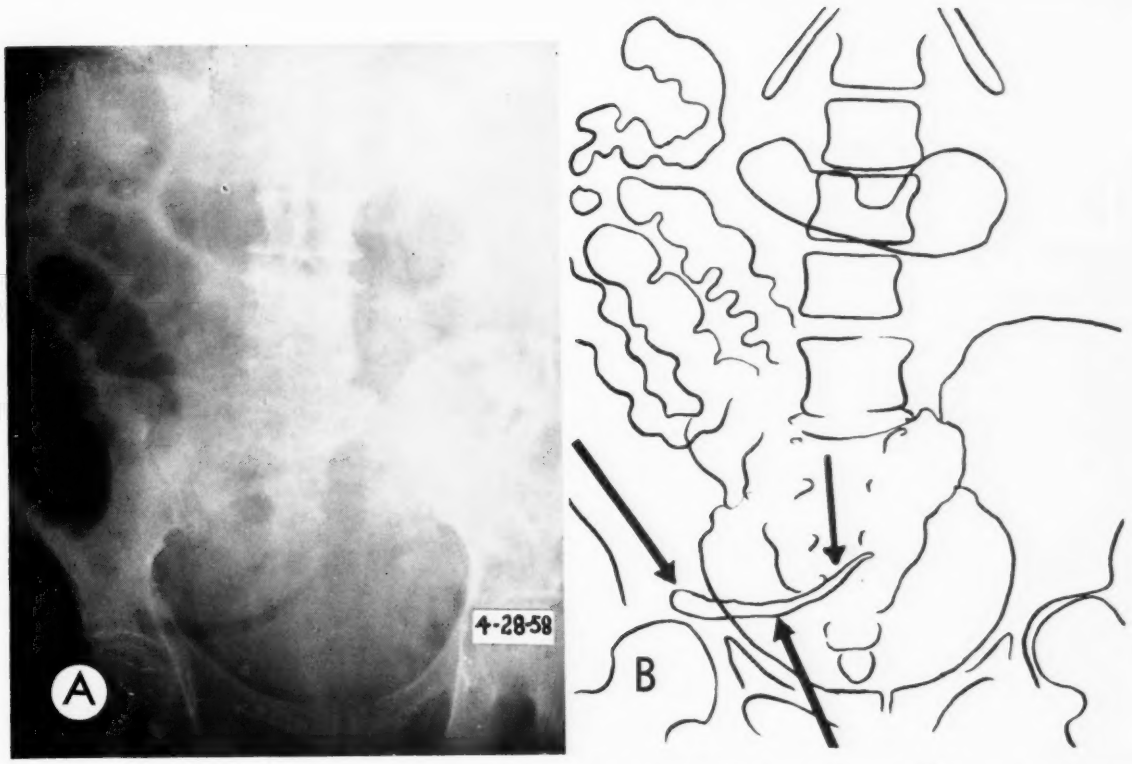

FIG. 2. (*A*) A narrow sickle-shaped collection of air is seen in the right lower abdomen. This roentgenogram was made in supine position. (*B*) Line drawing of *A*. See Figures 3 and 4 for roentgenograms made twenty-four hours later in the supine and right lateral decubitus positions.

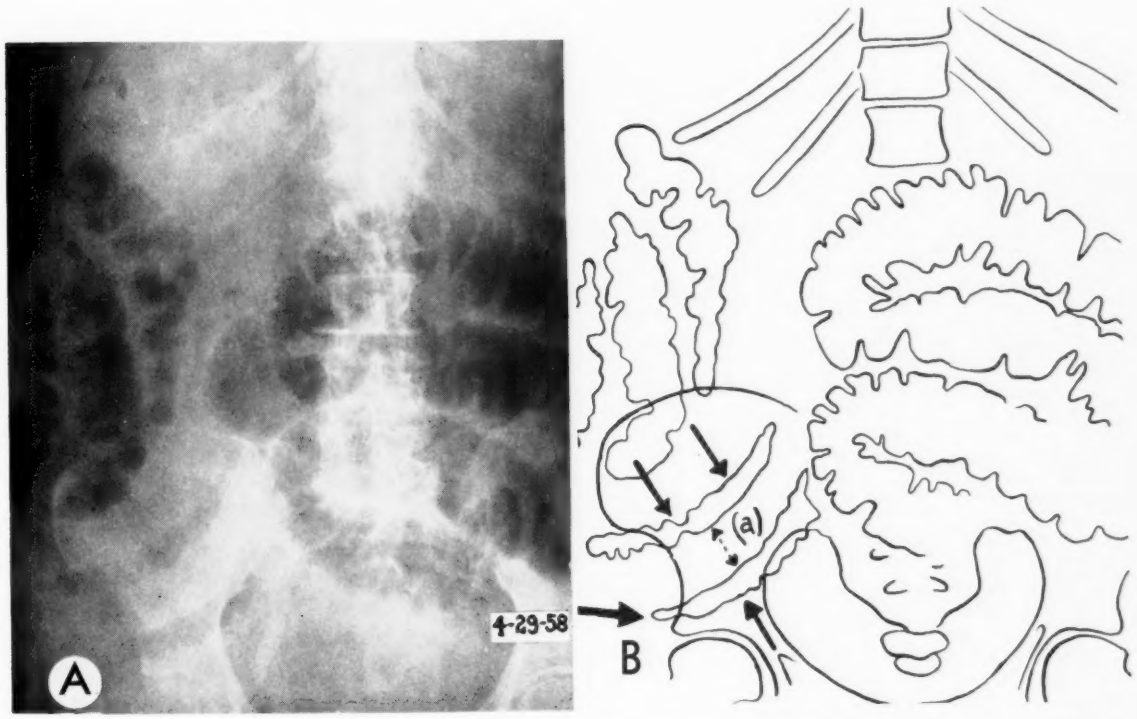

FIG. 3. (*A*) A roentgenogram made in supine position twenty-four hours after the supine roentgenogram shown in Figure 2 shows two sickle-shaped collections of gas in the right lower abdomen. Note the distance (a) between these collections of gas, which might be due to intraluminal fluid, extraluminal fluid between the loops, or to markedly edematous walls of two adjacent loops of intestine. Note this same area on the right lateral decubitus roentgenogram (Fig. 4). Note also the occurrence of distention of several loops of small bowel in the left mid-abdomen since the original examination made twenty-four hours earlier (Fig. 2). (*B*) Line drawing of *A*.

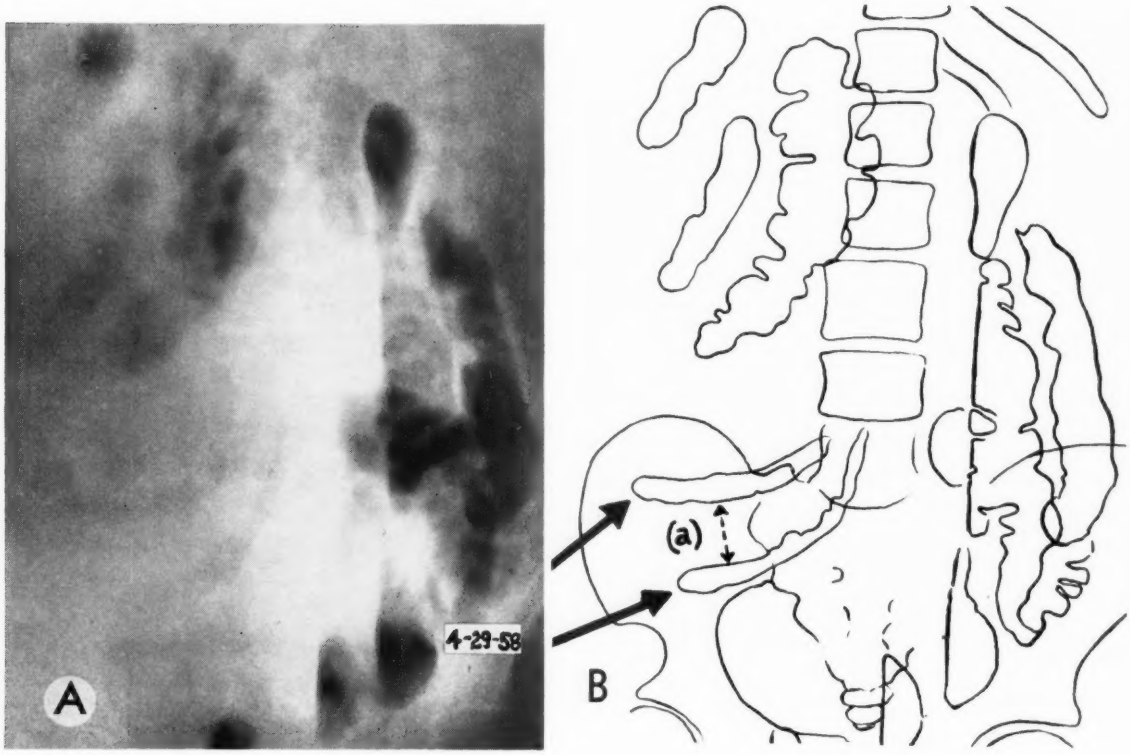

FIG. 4. (*A*) A right lateral decubitus roentgenogram barely shows the two sickle-shaped gas collections. Note that there has been very little change in the appearance of the water-density area (a) between the gas shadows. Furthermore, there has been no change in configuration of the sickle-shaped air shadows; *i.e.*, the air has not risen to the uppermost portions of these loops, and there is no evidence of fluid replacing the air in the lowermost portions of the loops, as occurred in the other dilated loops of bowel in the patient's left abdomen. Compare the behavior of the air and fluid in the dilated loops of bowel in the left abdomen with that in the supine roentgenogram (Fig. 3). The lack of any change in the width of the water-density area between the two sickle-shaped collections of air in the right lower abdomen led us to conclude that this was not intraluminal fluid or fluid between the loops, but rather due to thickening of the walls of two adjacent loops. (*B*) Line drawing of *A*.

of the aforementioned fluid-containing and air-containing portions of these loops. Such behavior was not noted with changes in position in the 2 patients here discussed (Fig. 3, *A* and *B*; 4, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B*). This caused us to conclude that these loops, although they contained primarily gas and apparently only minimal amounts of fluid, probably were very rigid and thick-walled, thus accounting for the lack of redistribution of the gas and fluid according to gravity when the position of the patient was changed. Also, the distance between these narrow intraluminal gas shadows of adjacent loops did not change when the position of the patient was changed (Fig. 3, *A* and *B*; and 4, *A* and *B*). This led us to conclude that this distance was due to markedly edematous walls of two adjacent loops, rather than to intraluminal fluid or intraperitoneal fluid between the loops.

In one patient we believed we were able to see the thickened bowel wall (Fig. 6, *A* and *B*, solid small black arrows in left lower quadrant) contrasted between the intraluminal gas and the extraluminal radiolucent shadow of mesenteric fat in the flank. Another roentgenologic sign of a rigid edematous loop was found in one of

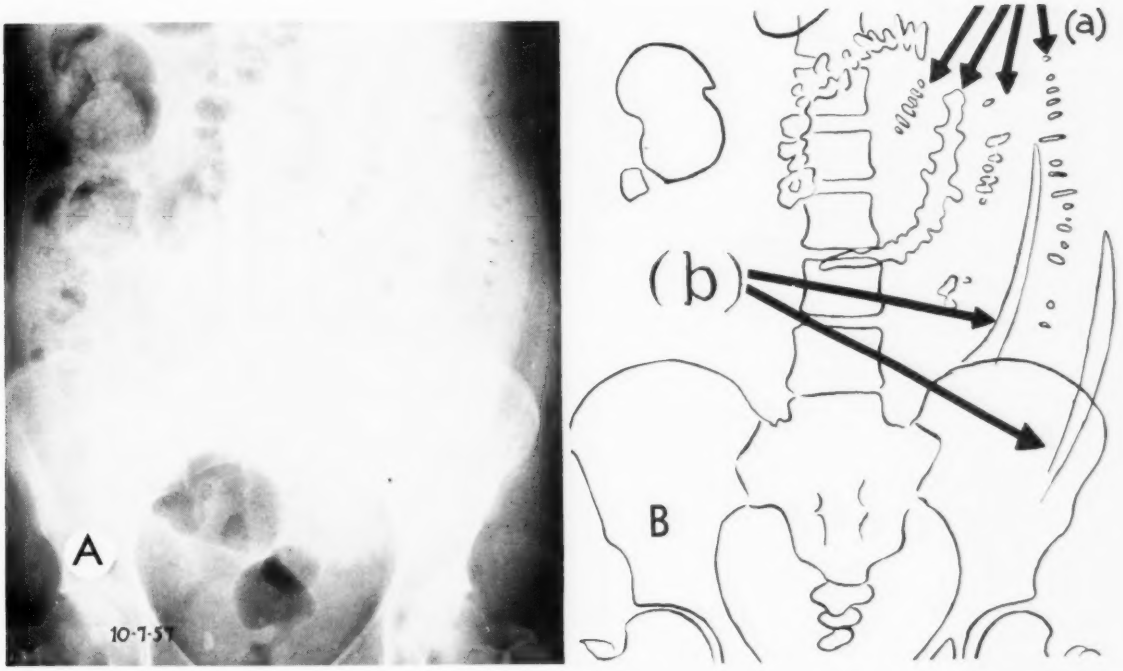

Fig. 5. (*A*) A roentgenogram made in supine position showing small collections of gas in what appear to be several fluid-filled loops of bowel in the left upper and mid-abdomen. These small collections of air are undoubtedly in the uppermost regions of the lumen between the mucosal folds. Furthermore, the mucosal folds must be rather markedly edematous, inasmuch as the water-density areas between the regularly spaced collections of gas are much wider than they would be if the folds were of normal width. (*B*) Line drawing of *A*. It is of interest that the omental fat in the left flank outlines the lateral margin of this abnormal loop of bowel, and apparently some omental fat vaguely outlines the area between the medial margin of this loop and the lateral margin of the adjacent loop as indicated by the large arrows (b). Compare these findings with the patient in supine position with those when the patient is in the right lateral decubitus position (Fig. 6).

the patients in whom the very wide spacing of the small collections of gas between the edematous mucosal folds could be seen in the involved loops on both the supine and decubitus roentgenograms (Fig. 5; *A* and *B*; and 6, *A* and *B*). On the decubitus roentgenogram the long, parallel, narrow gas-filled lumina of adjacent loops showed gas between the uppermost markedly edematous folds (Fig. 6, *A* and *B*). The spaces between the edematous folds in the most redundant wall of the involved loops were not at all apparent (Fig. 8, *A* and *B*), almost certainly due to the fact that small amounts of fluid were trapped between the folds, thus causing a more curvilinear appearance of the inferior portion of the air column in these loops (Fig. 6, *A* and *B*, small broken-line arrows) rather than a horizontal air-fluid level. Any excess fluid in the lumina of these loops probably descends by gravity into the redundant portions near the ends of these relatively straight rigid loops with small-caliber lumina. Thus, the air and fluid in these rigid loops did not act in a manner similar to that of air and fluid in ordinary cases of mechanical and paralytic ileus (Fig. 7, *A* and *B*).

The aforementioned findings were observed in 2 patients with proven thrombosis of the superior mesenteric vein. The explanation for the lack of redistribution of the gas in these loops when the position of the patient was changed is that probably the major content of the lumen of these

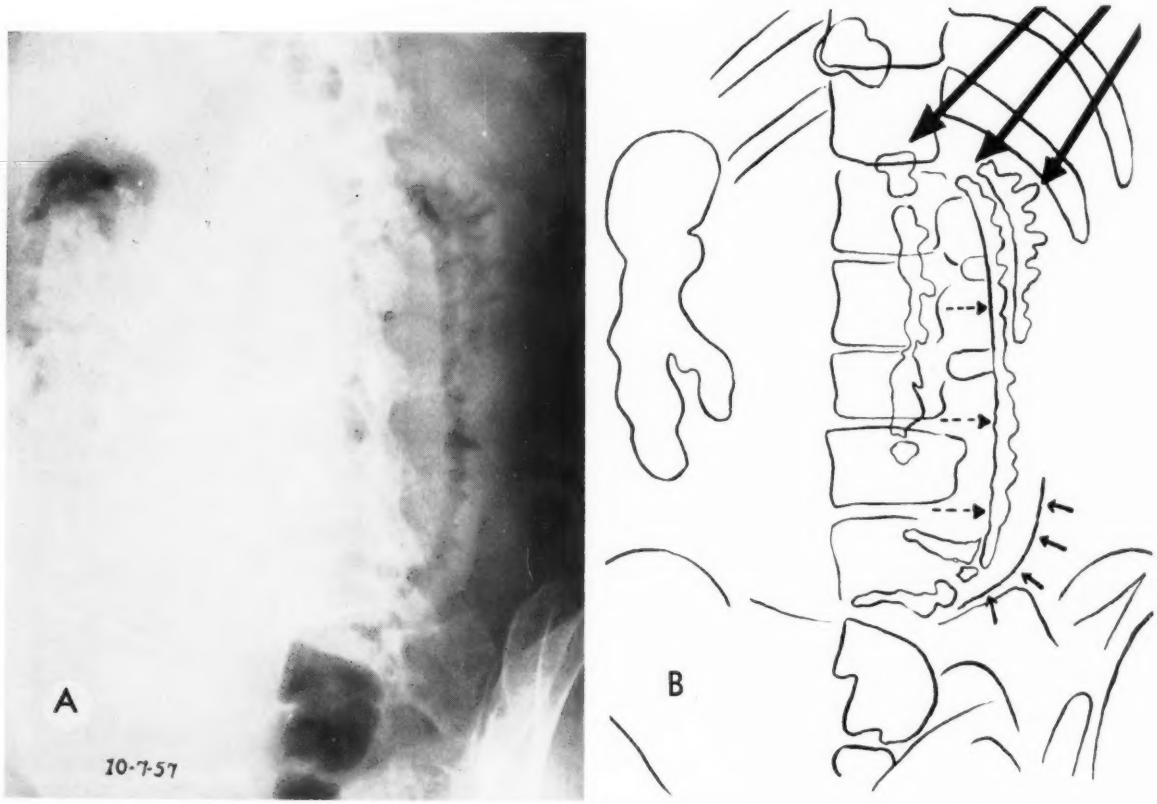

FIG. 6. (*A*) A roentgenogram made in right lateral decubitus position shows the absence of any detectable fluid levels in the abnormal loops seen in Figure 5. Furthermore, the markedly edematous mucosal folds in the uppermost portions of these loops are quite clearly outlined with small linear collections of air. Air does not outline mucosal folds in the most redundant portions of these rigid-appearing loops, apparently because of small amounts of fluid in the spaces between the mucosal folds. Note the left lateral margin of the most caudal portion of one of the loops (small solid arrows in left lower abdomen). The outer margin of the wall of this edematous loop can be seen by virtue of the omental fat, whereas the inner margin of the edematous wall is outlined by the intraluminal gas. (*B*) Line drawing of *A*.

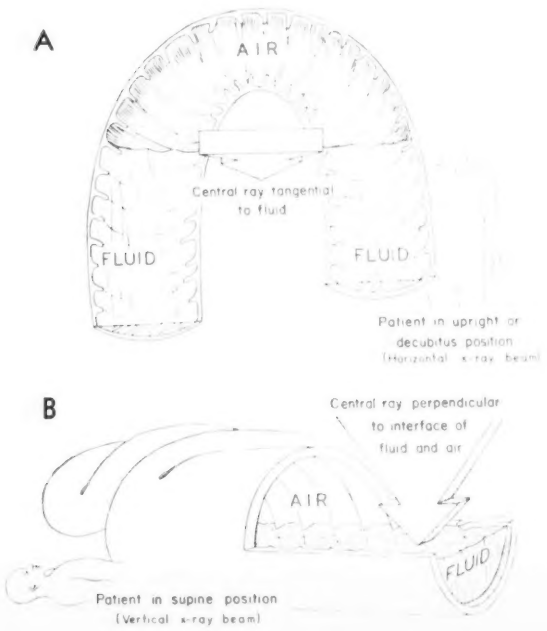

FIG. 7. Diagrams showing the behavior of pliable loops of intestine. *A* shows air in the uppermost portions of the loop and fluid in the most redundant portions of the loop. Thus, the interfaces between the air and fluid are easily seen manifested by air-fluid levels if the horizontal roentgen-ray beam is used. However, if the vertical roentgen-ray beam is used as in *B* the shadows of the air and fluid are frequently superimposed over each other, and the width of the lumen as seen on the roentgenogram will depend upon the relative amount of air and fluid within the lumen.

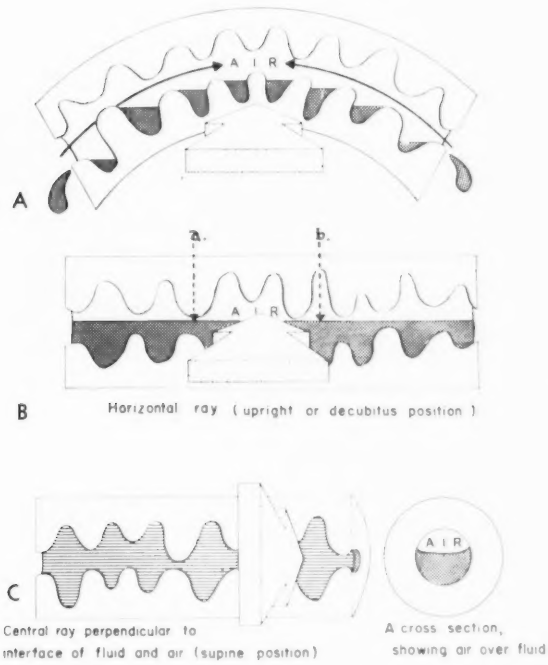

Fig. 8. Diagrams showing the behavior of rigid edematous loops. In *A* is shown the edematous bowel wall with markedly thickened mucosal folds. The air outlines the spaces between the edematous folds in the uppermost positions of the lumen, whereas small amounts of fluid fill in the spaces between the edematous folds in the lowermost portion of the lumen, but without any clear-cut evidence of any long air-fluid levels. Compare this figure with the appearance of the rigid loops seen in Figure 6. In *B* air and fluid are shown in a relatively narrow lumen due to edema of the bowel wall and marked thickening of the mucosal folds. The air and fluid would be clearly seen if the horizontal roentgen-ray beam were used. However, if a vertical roentgen-ray beam were used, no air would be shown in the lumen at (a), whereas a linear transverse collection of air would probably be seen at (b), where larger amounts of air had accumulated in the uppermost portions of the lumen between the mucosal folds. Furthermore, the distance between these small collections of gas should indicate the thickness of the folds. This should explain the appearance of the small collections of gas seen in Figure 5. In *C* is shown the appearance of the air column in a rigid edematous loop of intestine when a vertical roentgen-ray beam is used.

rigid edematous loops was gas, and that these rigid loops more or less trapped the gas and prevented its redistribution. Both of these patients had other nonspecific manifestations of ileus in bowel loops (Fig. 3, *A* and *B*; and 4, *A* and *B*), and at surgery there was marked edema of the involved loops of small bowel, but no evidence of any type of closed-loop obstruction to account for the bizarre behavior of the gas in the aforementioned rigid loops. Therefore, we believe that whenever such manifestations are seen, they should suggest the possibility of mesenteric venous thrombosis as the etiologic mechanism. Inflammatory diseases which produce edema of the bowel wall could conceivably produce this appearance on plain roentgenograms of the abdomen, although we have not seen any such cases to date.

SUMMARY

Although there is no pathognomonic roentgenologic sign of mesenteric vascular occlusion, the authors have described an additional observation on plain roentgenograms of the acute abdomen, which should suggest the possibility of mesenteric venous occlusion. The finding of a rigid, edematous segment of bowel in which a small relatively unspectacular collection of gas remains in a small, relatively straight or curvilinear lumen, and which does not change in distribution on upright or decubitus roentgenograms, is suggestive of an edematous segment of bowel due to mesenteric venous obstruction.

A brief summary of the etiology, pathophysiology, clinical findings, and the roentgen observations of other investigators has been given. It should be emphasized that the radiologist must be aware of this entity, and of the suggestive roentgenologic diagnostic criteria in order that he may properly consider the possibility of mesenteric vascular occlusions in the differential diagnosis of the acute abdomen, particularly in the elderly or cardiac patient.

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MESENTERIC VASCULAR DISEASE*

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THE mesenteric arteries are subject to the same atheromatous, thrombotic and embolic phenomena that are so frequently associated with the coronary and cerebral vessels. Coronary and cerebral vascular disease has been the subject of many studies, and the organic changes which result from their insufficiency or occlusion have been well explored and documented. Much has also been written about the clinical and pathologic aspects of acute mesenteric vascular occlusion,[2,6,9,15,16,17] but only in relatively recent times have some of the more subtle changes of mesenteric ischemia, insufficiency, and incomplete infarction become recognized. Since the main visceral organ supplied by the mesenteric arteries is the bowel, considerable information may be gained from study of plain roentgenograms of the abdomen, barium meal and enema examinations, as well as from contrast visualization of the aorta and its branches.

For some years it has been said that mesenteric artery abnormalities exhibit roentgenographically a specific pattern. It is presently known that this belief is far from conclusive. There are, however, certain roentgenographic manifestations which are encountered in mesenteric vascular occlusion, and have not been adequately stressed. The purpose of this paper is to demonstrate and discuss some of these manifestations which were observed during the past ten years at the Massachusetts General Hospital.

ETIOLOGY

The etiology of mesenteric vascular occlusion varies. Underlying systemic disease, generalized atherosclerosis, cardiac disease with endocardial vegetations, atrial fibrillation, recent myocardial infarction, and vasculitis particularly play important roles. Blood dyscrasias, such as polycythemia and leukemia, and cardiac and abdominal surgical procedures may also be responsible for its occurrence. In some instances no apparrent cause is demonstrable even at autopsy, and the occlusion may be due to injury to the vessel walls.

Mesenteric arterial occlusion may be either embolic or thrombotic in nature, and it is much more serious than venous occlusion which is always thrombotic and frequently due to disease of the splenoportohepatic system and infection of the visceral and pelvic organs.

CLINICAL FINDINGS

Pain is usually, but not always, a prominent feature in the patient presenting with mesenteric vascular disease. The variable types of pain which may occur often reflect the kind or extent of vascular involvement but are not necessarily pathognomonic. In acute mesenteric occlusion there is often a sudden onset of excruciating abdominal pain associated with varying degrees of shock. The stools and vomitus may contain blood. In the late stage the entire picture is further complicated by peritonitis and infection. In subacute or chronic vascular occlusion, pain may be described as dull or aching and is often periumbilical. It may be anginal in that attacks occur spasmodically and may last for minutes or hours. A striking feature in some patients has been the relation of pain to meals, appearing one to two hours after eating and lasting two to four hours. The severity of this postprandial pain may be proportional to the quantity of food ingested, presumably a reflection of work overload on the relatively ischemic bowel. In chronic cases the symptoms may be very mild; not infrequently a

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week or more may elapse before the true nature of the lesion is suspected.

Anorexia and nausea are frequent. Vomiting may occur three to four times daily in the subacute cases. Vomitus will contain blood only in the acute case. Watery diarrhea, sometimes "soap bubbly" in appearance, may accompany the vomiting. Guaiac positive stools or frank blood will be present if the bowel is infarcted.

Temperature elevation up to 102° F. and white blood cell counts of 15,000 to 20,000 are not uncommon. Microscopic and macroscopic hematuria may indicate associated renal vascular ischemia. Elevated alkaline phosphatase may suggest that the celiac axis is also involved. In the cases of chronic mesenteric insufficiency, malabsorption syndromes may be documented by various studies of which the d-xylose test seems to be the most sensitive.[1]

PROGNOSIS

The prognosis of acute mesenteric occlusive disease in general is poor and it is influenced by the extent and rapidity of the process, its prompt recognition, and its resectability. A mortality ranging from 60 to 85 per cent[2,7,9] has been recorded in those in whom surgical resection was attempted. Spontaneous recovery from mesenteric occlusion, however, does occur,[2] but is exceptional. In subacute and chronic cases, the prognosis is uncertain, as the disease often may not be recognized.

PHYSIOPATHOLOGIC CONSIDERATIONS

To better understand the changes which may occur clinically, pathologically, and roentgenographically, it may be worthwhile to formulate a pattern of the findings which can be anticipated in the bowel as a result of mesenteric vascular occlusion of varying degree and extent.

Angina from coronary insufficiency and cerebral changes due to multiple "small strokes" are accepted clinical entities. Mesenteric insufficiency and angina similar in nature[7,8] are frequently unrecognized clinical entities. Actually, there may be a much larger incidence of misunderstood border-line mesenteric artery insufficiency, as the mesenteric arteries seem to be subject to the same atheromatous, embolic, and thrombotic phenomena seen in other vessels of similar size. However, most vascular disease is really manifest in the patient by the secondary physiologic, chemical and pathologic changes which occur in the organ receiving the vascular supply.

Since the mesenteric vessels principally supply the bowel, secondary changes from their occlusion may be subtle and chronic, or dramatically acute and fatal.

When the arterial blood supply to the bowel is cut off completely by either thrombi or emboli, the result in the intestine is infarction. The congested bowel and its mucous membranes become purplish-red, thickened and edematous, and finally hemorrhage and necrosis of the wall supervene. Similar changes are encountered in the involved mesentery, and not infrequently the peritoneal cavity contains malodorous bloody fluid.

If the blood flow through the bowel is partially reduced to a point less than that required to produce actual infarction, it is theoretically conceivable that a state of incomplete bowel function or bowel decompensation might occur. The digestive processes and absorption through the lacteals may be less than normal. That such a state has occurred has been observed clinically in a few patients, resulting in sprue-like malabsorption syndromes.[14] Further, interference with blood flow in mesenteric veins may result in intestinal edema and functional "congestive failure," and such secondary changes may continue for weeks or even months.[3]

In the bowel there is another feature to be considered. That is the fact that the normal bowel contains an abundant and variable supply of bacteria. Therefore, if ischemia is of sufficient degree to interfere with the integrity of the mucosa, inflam-

matory changes in the bowel wall are likely to be more severe than relatively aseptic ischemia or infarction in other organs such as the heart or brain. Thus, some cases, either with or without malabsorption syndrome presumed to be on a vascular basis, have also exhibited localized areas of severe inflammation with narrowing of the lumen, mimicking regional enteritis. It is interesting to note that more than a few instances of "regional enteritis" do not have all the pathologic criteria of Crohn's disease. It is conceivable that many of these cases may have a vascular etiology. Some pathologists state that the changes in some cases of regional enteritis are the same as those which might be expected and have been observed in localized occlusion of branches of the mesenteric arteries. The pathologic interpretation is difficult in many cases because the entire vascular supply is not available for study, and in some specimens primary arterial and venous changes are obscured by the overriding inflammatory changes.

Acute, complete infarction of a segment of bowel will sometimes result in inflammatory thickening of the intestinal wall and its surrounding fat to an extent sufficient to produce a mass which may be palpable or visible on roentgenograms. Such a mass may simulate a tumor, but the mucosa appears to be grossly intact.

When ischemia is temporary and localized, infarction is usually incomplete. In such an instance a localized contracted segment of bowel simulating ulcerative colitis may occur, and the patient may recover without further incident.

If a localized vascular ulcerative colitis or regional enteritis is of severe degree and the patient recovers, secondary stricture of the bowel may result.[11] In one such healed case it is interesting to note that at operation the external wall of the bowel appeared to be of the same caliber throughout, although the lumen was markedly narrowed.

ROENTGEN MANIFESTATIONS

The roentgen manifestations of mesenteric vascular disease depend on the rapidity and extent of involvement, the size of the vessels involved, the precise anatomic site of narrowing or occlusion, the extent of available collateral circulation, the integrity of the bowel mucosa, and the presence or absence of bacterial infection of the bowel.

Very little has been published about the roentgen aspect of this disease. The classic appearance of acute superior mesenteric artery occlusion as seen on plain roentgenograms and described by Rendich and Harrington[10] in 1944, by Harrington[5] in 1947, and by Sante[12] in 1951 is that of dilated small and large bowel with a sudden stoppage of the air-filled column in the transverse colon near the splenic flexure; in our experience this is rare.

Cases of arterial insufficiency producing "mesenteric angina" may show little or no change on plain or barium studies (Case I). Likewise, extensive infarction may or may not produce gaseous distention of the bowel. When it does, the gas-distended bowel does not usually follow the involved anatomic vascular pattern. Of 18 cases of proved superior mesenteric artery occlusion, 13 patients showed the presence of a nonspecific, bizarre pattern of ileus of various degree, and in only 3 cases the so-called typical roentgenographic findings were observed (Case II). In 2 instances the gas pattern was considered normal. Of 16 superior mesenteric vein occlusions, 12 cases presented findings quite similar to those of arterial occlusion, chiefly nonspecific ileus; in 2 instances, there was progressive ascites obscuring the intestinal gaseous pattern. In 2 cases with superior mesenteric vein thrombosis, the infarcted bowel, as revealed on the plain roentgenograms, showed marked thickening of the walls and the segments of gas-filled loops paralleled each other, similar to a cluster of bananas, with fixation of the loops of bowel (Case IV and Case VII). From the present studies, the differentiation of mesenteric artery occlusion from vein occlusion may be extremely difficult in many

instances. There were only 2 cases of inferior mesenteric occlusion which likewise showed scattered gas in the bowel without any specific diagnostic abnormality.

CONTRAST STUDIES IN VASCULAR OCCLUSIVE DISEASE

Emphasis was recently placed on the value of contrast studies in the diagnosis of mesenteric vascular disease, particularly in the subacute and chronic forms.[4,18] This type of examination may be quite feasible, often informative and not necessarily hazardous. In 15 patients who had such investigation, the following findings were demonstrated: In cases where the classic appearance of superior mesenteric occlusion simulated large bowel obstruction near the splenic flexure, findings from a barium enema examination revealed the true nature of the disease (Case III). In acute mesenteric vascular occlusion, barium meal studies revealed marked dilatation of the bowel with edema of the bowel wall and increased thickness between the barium filled loops of the bowel (Case IV). The mucosal folds were quite swollen and at times widely spaced. The bowel gas pattern remained unchanged for a considerable period of time because of ileus of the infarcted intestine.

There is definite evidence that some gastrointestinal malabsorption syndromes are vascular in origin. In these cases barium studies of the small bowel may reveal dilated loops and mucosal thickening, with flocculation and segmentation of the barium column presenting a deficiency pattern (Case V). When a long segment of the bowel is relatively ischemic, malabsorption syndrome may or may not be present, and the contrast substance may outline a narrowed segment of the bowel with some degree of fixation. These ribbon-like segments of various lengths may be smooth or at times show multiple punctate ulcerations, simulating regional ileitis and ulcerative colitis. The involved segments often show smooth tapering ends continuous with the normal bowel which may or may not be dilated (Case VI and Case VII). In localized bowel infarction, the mucosal folds may be markedly swollen with inflammatory infiltration of the periepiploic fat about the infarct, and the appearance may mimic an intraluminal tumor (Case VIII). Localized stricture of the bowel following recovery from ischemia and infarction occurs and often shows smooth tapering ends without overhanging edges. On the other hand, a small localized infarction may heal without any residual evidence if the collateral circulation has developed adequately.

REPORT OF CASES

Case 1. *Superior Mesenteric Artery Occlusion with Slight Roentgenographically Demonstrable Abnormalities* (Fig. 1, *A* and *B*). A fifty-seven year old male was admitted to the hospital because of a 30 pound weight loss and pain in the upper abdomen. The pain was described as periumbilical and colicky in nature, occurring half-hour after eating and lasting one to two hours, accompanied by nausea but no vomiting. One month later exploratory laparotomy revealed slight chronic inflammatory reaction with dilated capillaries and lymphatics in the serosa of the small bowel. Biopsy of the mesenteric lymph node, jejunum and liver, however, showed no diagnostic abnormality, and a diagnosis of Whipple's disease was unwarranted pathologically. The patient was discharged home on metacorten with resultant improvement of symptoms.

A year later a repeat gastrointestinal series showed no gross abnormality in the stomach and only slight thickening of the mucosal folds in the small bowel. Shortly after the gastrointestinal series, aortography was performed and demonstrated an apparent obstruction of the superior mesenteric artery with subsequent filling of some of its branches from below via anastomosis of the left colic and middle colic arteries. Two days later thromboendarterectomy of the superior mesenteric artery was carried out with resection of the terminal 2 or 3 feet of the frankly infarcted ileum. The patient recovered from the procedure and was discharged from the hospital three weeks postoperatively.

He was re-admitted, however, ten days later with symptoms of abdominal cramps and persistent leukocytosis. At that time d-xylose absorption test showed evidence of malabsorption

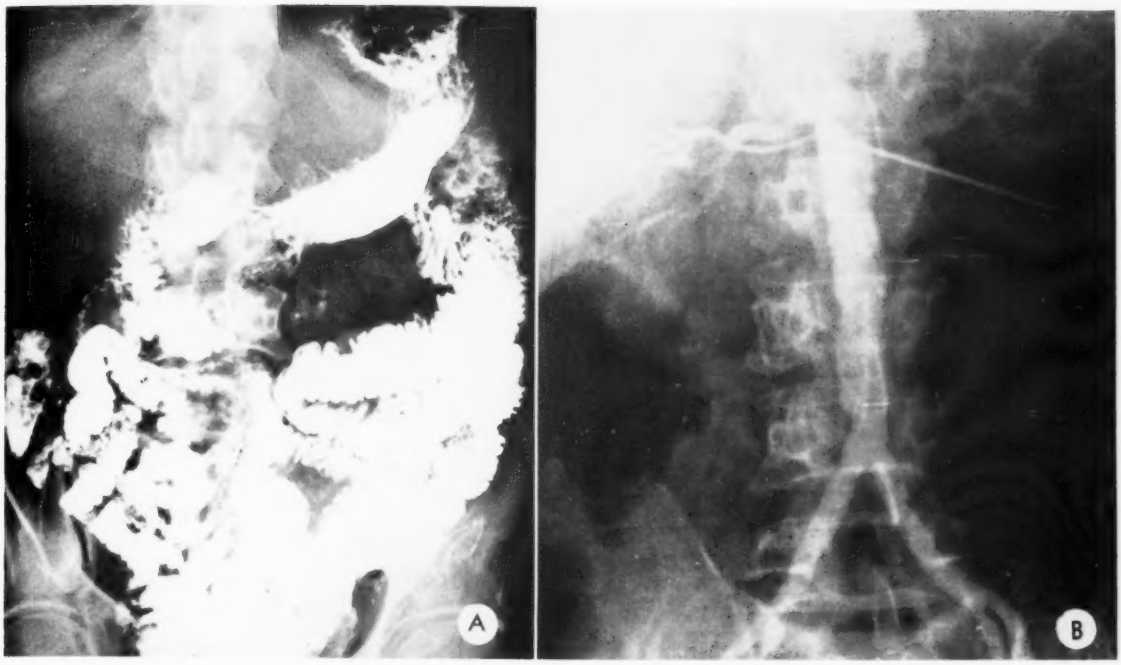

Fig. 1. Case I. (*A*) Barium meal study shows slight coarsening of the mucosal folds. (*B*) Abdominal aortogram demonstrates complete occlusion of the superior mesenteric artery and atherosclerosis of the abdominal aorta. Collateral circulation is evident (not well demonstrated in reproduction).

syndrome. He was discharged home on a low fat diet.

It is interesting to note that an apparent obstruction of the superior mesenteric artery produced very little change in the appearance of the small bowel on plain roentgenograms or by contrast study.

Case II. *Mesenteric Artery Thrombosis: Plain Film Manifestation* (Fig. 2). A sixty-two year old female was admitted because of pallor, pain and numbness of both legs of five days' duration. She was found to have aortic, iliac, femoral and popliteal thromboses and embolectomies were performed.

Preoperative roentgenograms of the abdomen showed gas-filled loops of small and large bowel, rather nonspecific in nature, scattered in the abdomen.

Three days after operation the patient developed abdominal pain, fever, chills, elevated white blood cell count, 2 plus guaiac stool with abdominal distention and diminished peristalsis. A roentgenogram of the abdomen at that time showed definite distention of some loops of the small and large bowel. There was marked distention of the proximal colon to the region of the splenic flexure. The descending colon was collapsed. Repeat roentgenograms two days later showed considerable change of the gas pattern with a few loops of gas dilated small bowel in the left mid-abdomen. The gas in the proximal colon was mostly evacuated. The cecum, however, was somewhat contracted and there was a polypoid soft tissue filling defect projecting from its medial aspect. These changes were thought to be quite suggestive of mesenteric thrombosis.

The patient was explored and gangrenous bowel from the lower jejunum to the hepatic flexure of the colon was found. A resection was performed.

The patient died one week later and postmortem examination showed occlusion of the superior mesenteric artery with an aortic mural thrombus.

Case III. *Superior Mesenteric Vein Occlusion Simulating Large Bowel Obstruction at the Splenic Flexure* (Fig. 3, *A*, *B* and *C*). A fifty-nine year old male came into the hospital because of dysphagia of three months' duration. Roentgen examination showed a constricting lesion in the lower third of the esophagus. Esophagectomy was performed and revealed a benign esophageal stricture with peptic ulcera-

tion. One week after the operation the patient had a sudden onset of abdominal distention associated with pain. There was good intestinal peristalsis. He had a few bowel movements and passed gas per rectum. The etiology of his abdominal distention was obscure. There were 4 plus guaiac stools and a white blood cell count of 11,400.

Roentgenograms showed a large amount of gas in the small and large bowel.

Approximately one month after operation, his condition took a sudden downhill course and he expired.

A plain roentgenogram taken before death showed marked dilatation of the small and large bowel down to the region of the splenic flexure. The descending colon was contracted. Barium enema study, however, demonstrated no colonic obstruction, and the descending colon was less dilated than the proximal colon. These changes were quite compatible with superior mesenteric vein occlusion, which was found at postmortem examination of this patient.

CASE IV. *Acute Superior Mesenteric Vein Thrombosis* (Fig. 4, *A* and *B*). A forty-nine year old man was admitted to the hospital with a four day history of vague upper abdominal pain. While he was in the emergency ward, he had a small, bright red bloody stool. A Levin tube was inserted into the stomach approximately two hours later and revealed bloody gastric contents. While he was in the hospital he had several blooody stools. Repeated blood transfusions failed to bring the patient out of shock. He had no previous history of peptic ulcer, anorexia, nausea, or vomiting. Physical examination showed a distended abdomen with diffuse epigastric tenderness and occasional high-pitched rush of peristalsis.

A plain roentgenogram of the abdomen revealed diffuse haziness suggesting ascites. There were a few gas-filled loops of small bowel in the right mid-abdomen which paralleled and were separated from each other for a distance of 2 cm., either due to markedly thickened bowel wall or fluid accumulating between the loops. A few gaseous loops were also noted in the left flank.

An emergency gastrointestinal series showed an unusually widened esophagus and a gas distended stomach. There was no active peristalsis. The stomach and the duodenal bulb were

FIG. 2. Case II. Plain roentgenogram of the abdomen. A large amount of gas is seen in the small bowel, ascending and transverse colon to the splenic flexure. The descending colon contains no significant amount of gas.

free of ulceration. The barium went through the pylorus into the duodenum and jejunum in an unusually rapid fashion by gravity, and the upper small bowel was markedly dilated. The mucosal folds in the jejunum were prominent and edematous. There was marked delay in passage of the barium through the small bowel.

At operation a superior mesenteric vein thrombosis with infarction of the entire small bowel and its mesentery was found. No resection was possible, and the patient expired seven hours postoperatively.

Postmortem examination confirmed the operative findings.

CASE V.* *Mesenteric Vascular Disease with Malabsorption Syndrome and "Regional Enteritis" of the Terminal Ileum* (Fig. 5, *A* and *B*). A sixty-four year old male was admitted into the hospital with a history of diarrhea of two years' duration, a 50 pound weight loss in nine months, and weakness of several months' duration. He had had a myocardial infarction eight

* Cabot Case 43491. *New England J. Med.*, 1957, *257*, 1130–1135.

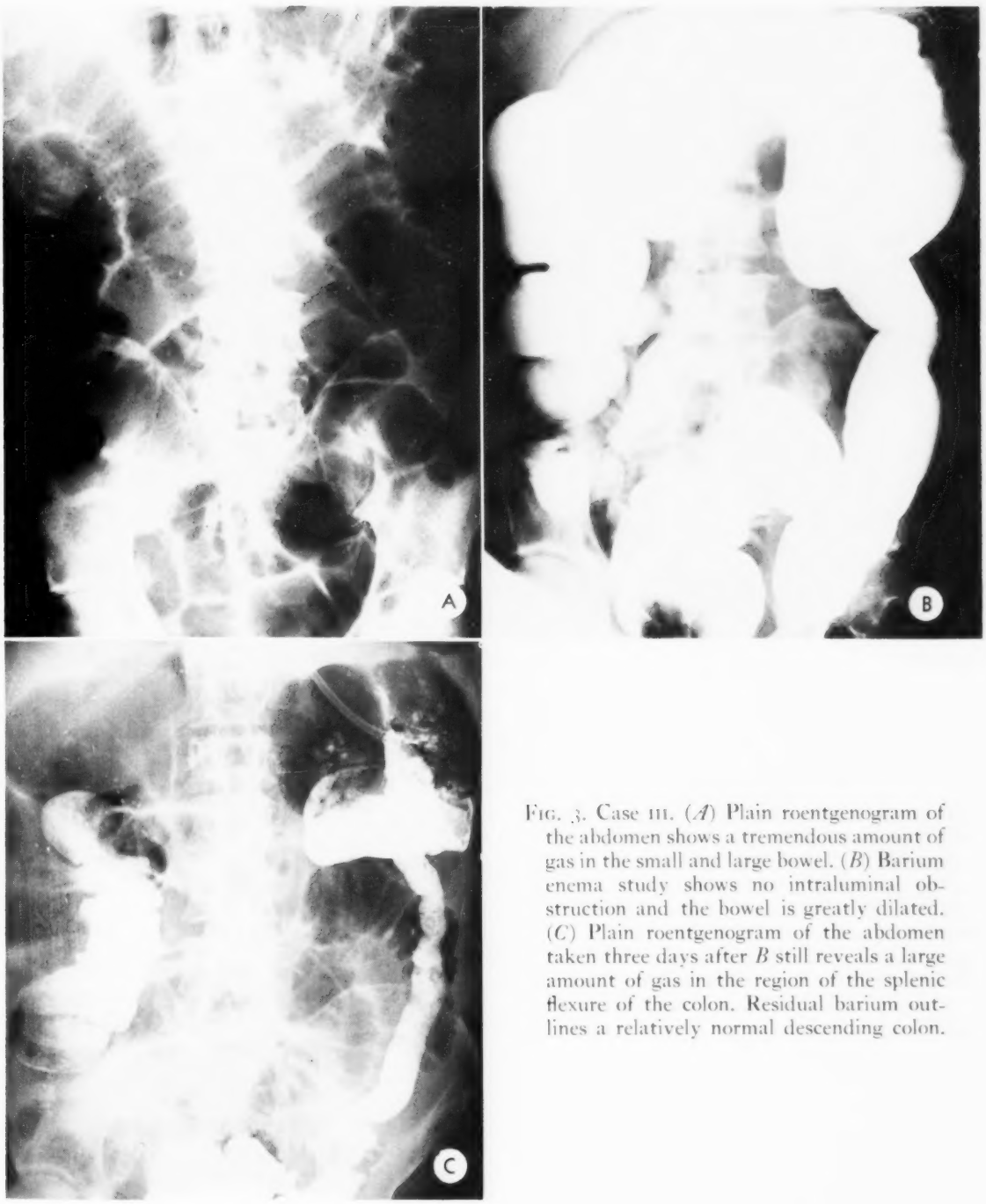

Fig. 3. Case III. (*A*) Plain roentgenogram of the abdomen shows a tremendous amount of gas in the small and large bowel. (*B*) Barium enema study shows no intraluminal obstruction and the bowel is greatly dilated. (*C*) Plain roentgenogram of the abdomen taken three days after *B* still reveals a large amount of gas in the region of the splenic flexure of the colon. Residual barium outlines a relatively normal descending colon.

years before entry. He had noted insidious onset of diarrhea without blood, averaging 3 to 4 stools per day, two years prior to admission. Abdominal pain, vomiting and anorexia were noted for about one month. A small bowel series showed moderate dilatation of the bowel throughout, with slight thickening of the mucosal folds. There was some irregularity of the mucosa of one loop of the terminal ileum. Some degree of flocculation and segmentation of the barium column was also noted.

Of the absorption tests carried out, only the

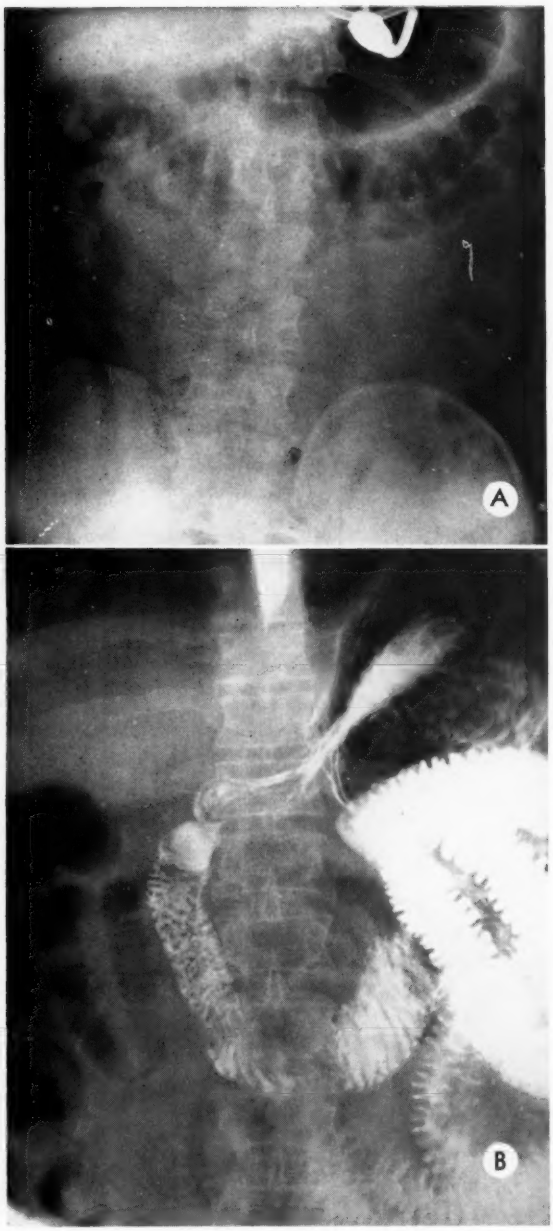

FIG. 4. Case IV. (*A*) Plain roentgenogram of the abdomen shows evidence of ascites, and a few peculiar gas shadows suggesting thickened intestinal walls in the right mid-abdomen. (*B*) Barium meal study shows markedly edematous mucosal folds in the upper small bowel. The jejunum is considerably dilated.

d-xylose test was found to be abnormal. The hospital course was marked by a temperature elevation of 103° F. and 104° F. Because of a probable diagnosis of lymphoma in mind, the patient was given a course of nitrogen mustard therapy empirically without satisfactory response. An exploratory laparotomy was performed and revealed 4 feet of edematous terminal ileum with thickened areas at the mesenteric border. There were enlarged mesenteric lymph nodes which showed only acute and chronic inflammation by biopsy. He later developed pneumonia and expired one month postoperatively

Postmortem examination showed that the 3 feet of terminal ileum were indurated and thickened. The overlying peritoneal surfaces were considerably thickened with numerous adhesions. The lesion in the terminal ileum was sharply demarcated with edematous mesentery. The mesenteric vessels showed considerable atherosclerosis but no embolus or thrombus was found. It was believed that the findings in the small bowel could be the result of ischemia due to vascular insufficiency. The changes were not those commonly seen in regional enteritis due to the lack of focal granulomas and giant cell systems.

CASE VI. *Superior Mesenteric Artery Embolus with Secondary Malabsorption Syndrome and Multiple Areas of "Regional Enteritis"* (Fig. 6, *A*, *B* and *C*). A fifty-four year old female was admitted to the hospital because of epigastric pain with repeated vomiting and rectal bleeding of two days' duration. Five days before entry she had had a right cerebral embolus resulting in left hemiparesis and thick speech. On admission, the white blood cell count was 16,000.

Approximately ten hours after admission, a laparotomy was performed which revealed a blue-gray small bowel. The large bowel had good pulses. It was felt that she had a superior mesenteric artery embolus. The superior mesenteric artery was explored and an embolus was removed. No bowel resection was done.

Postoperatively, the patient continued to have loose bowel movements daily, and it was a difficult problem to maintain her nutrition.

Approximately six weeks after operation, cheilosis, a beefy tongue, and the appearance of the skin suggested protein deficiency, and the patient seemed to be developing a sprue-like syndrome. Small bowel series done approximately two months after operation showed widespread abnormality of the small bowel characterized by multiple areas of ribbon-like

Fig. 5. Case v. (*A*) Barium meal study shows some dilatation of the small bowel. (*B*) Roentgenogram of the abdomen taken three hours after *A* shows flocculation of the barium column. There is an irregularity of the walls of a segment of the terminal ileum in the right abdomen.

narrowing and segmentation with loss of normal mucosal pattern. During subsequent examinations of the small bowel in a period of two months, these changes had regressed considerably, and the last examination showed some return of normal mucosal pattern with continuity of the barium column in the small bowel.

A cerebral embolus incapacitated the patient and nutrition became the chief problem. Her diarrhea, however, had ceased in about six months.

Case vii. *Thrombosis of the Superior Mesenteric Vein Producing "Ileitis" Deformity of the Small Bowel* (Fig. 7, *A* and *B*). A fifty-one year old male with a questionable history of angina entered the hospital with abdominal pain of one week's duration. The pain was not related to food nor did it radiate to the back or shoulder. No nausea, vomiting or fever were noted. On admission the white blood cell count was 16,000, and there was no intestinal peristalsis on auscultation. While the patient was in the hospital, he began to bleed from the rectum and died twelve days after entry.

Plain roentgenograms of the abdomen showed the presence of gas in the proximal colon. There were three peculiar gas shadows in the right mid-abdomen paralleling each other and suggesting stiff gas-filled loops of small bowel with thickened walls.

Barium enema study with reflux into the ileum showed markedly narrowed loops of terminal ileum with edematous folds. The ribbon-like narrowing showed some serration of its wall, simulating regional ileitis.

At autopsy the small bowel was gangrenous due to mesenteric vein thrombosis. The roentgenographic appearance of this case is quite similar to that of regional enteritis, but the differential diagnosis lies in the clinical history.

Case viii.* *Inferior Mesenteric Occlusion*

* Cabot Case 36081. *New England J. Med.*, 1950, *242*, 294–298.

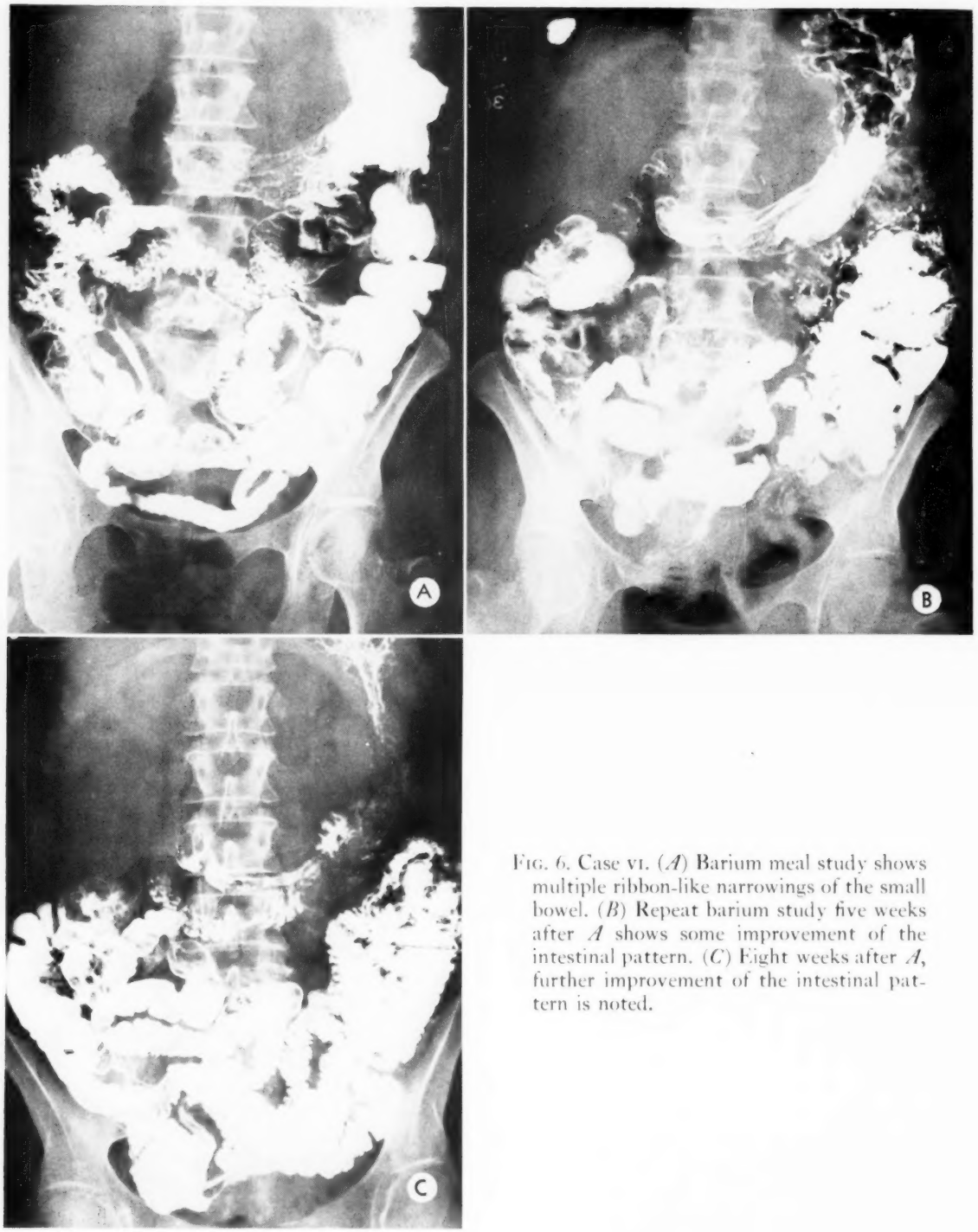

Fig. 6. Case VI. (*A*) Barium meal study shows multiple ribbon-like narrowings of the small bowel. (*B*) Repeat barium study five weeks after *A* shows some improvement of the intestinal pattern. (*C*) Eight weeks after *A*, further improvement of the intestinal pattern is noted.

Producing Intraluminal Pseudotumor (Fig. 8, *A*, *B* and *C*). A sixty-one year old male had had intermittent claudication with clinical absence of pulsation in the feet for about ten years. He also had had duodenal ulcer and gastritis. Exacerbation of these symptoms and partial pyloric obstruction prompted his entry into the hospital. Sixteen months prior to entry he had a posterior myocardial infarction. While he was in the hospital receiving conservative therapy

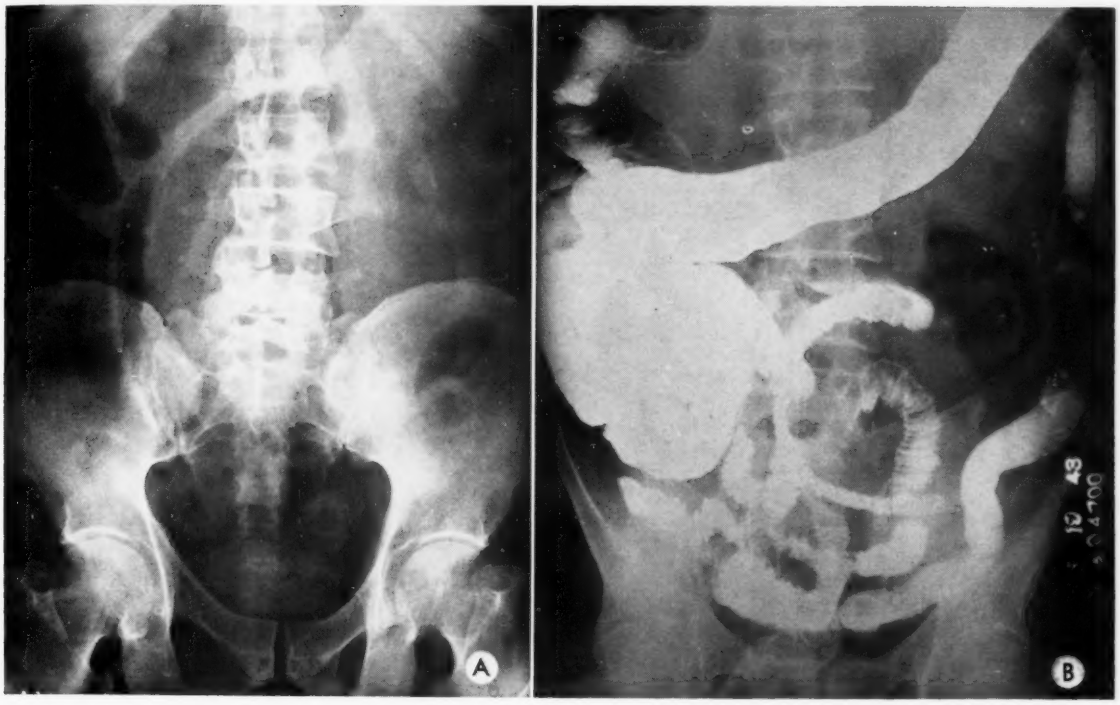

Fig. 7. Case VII. (*A*) Plain roentgenogram of the abdomen shows some gas in the large and small bowel. There are 3 loops of small bowel in the right mid-abdomen. (*B*) Barium enema study with reflux into the ileum shows a long segment of ribbon-like narrowing of the terminal ileum with superficial ulcerations.

for ulcer, he developed left lower quadrant pain with elevation of fever to 102° F. and a white blood cell count of 15,500.

A barium enema examination revealed an area of narrowing in the descending colon with a large amount of fecal matter just proximal to it. One observer stated that fluoroscopically this narrowed area could be distended to almost normal size. Within this area of abnormality there were multiple lobulated filling defects. The bowel wall of the rectosigmoid region was serrated, suggesting multiple small ulcerations.

A repeat barium enema study performed one day later showed no great change. The patient developed a complete occlusion of the aorta and died of acute left heart failure.

Postmortem examination showed the changes in the descending colon and sigmoid to be due to local gangrene and inflammatory reaction in the pericolic fat which in turn were due to small peripheral emboli. There was an aortic thrombus occluding the inferior mesenteric artery completely. The small ulcerations in the rectosigmoid colon were the result of thrombosis of the terminal arteries.

DISCUSSION

As is the case in all roentgen diagnoses, the clinical history is essential to the proper interpretation of the roentgenographic findings. Roentgen manifestations alone are rarely sufficient to reveal the specific diagnosis, but in the presence of the following findings, mesenteric vascular occlusion should always be suspected:

1. Acute abdominal pain, vomiting, and varying degrees of shock in a patient presenting possible etiologic factors for mesenteric vascular disease such as generalized atherosclerosis, atrial fibrillation, myocardial infarction, liver disease, blood dyscrasia, and antecedent aortic or abdominal surgery. The plain roentgenograms may reveal (a) nonspecific ileus, or uncommonly in acute superior mesenteric artery occlusion, the "classic" pattern of gas-dilated small and large bowel to the region of the splenic flexure; (b) signs of fixa-

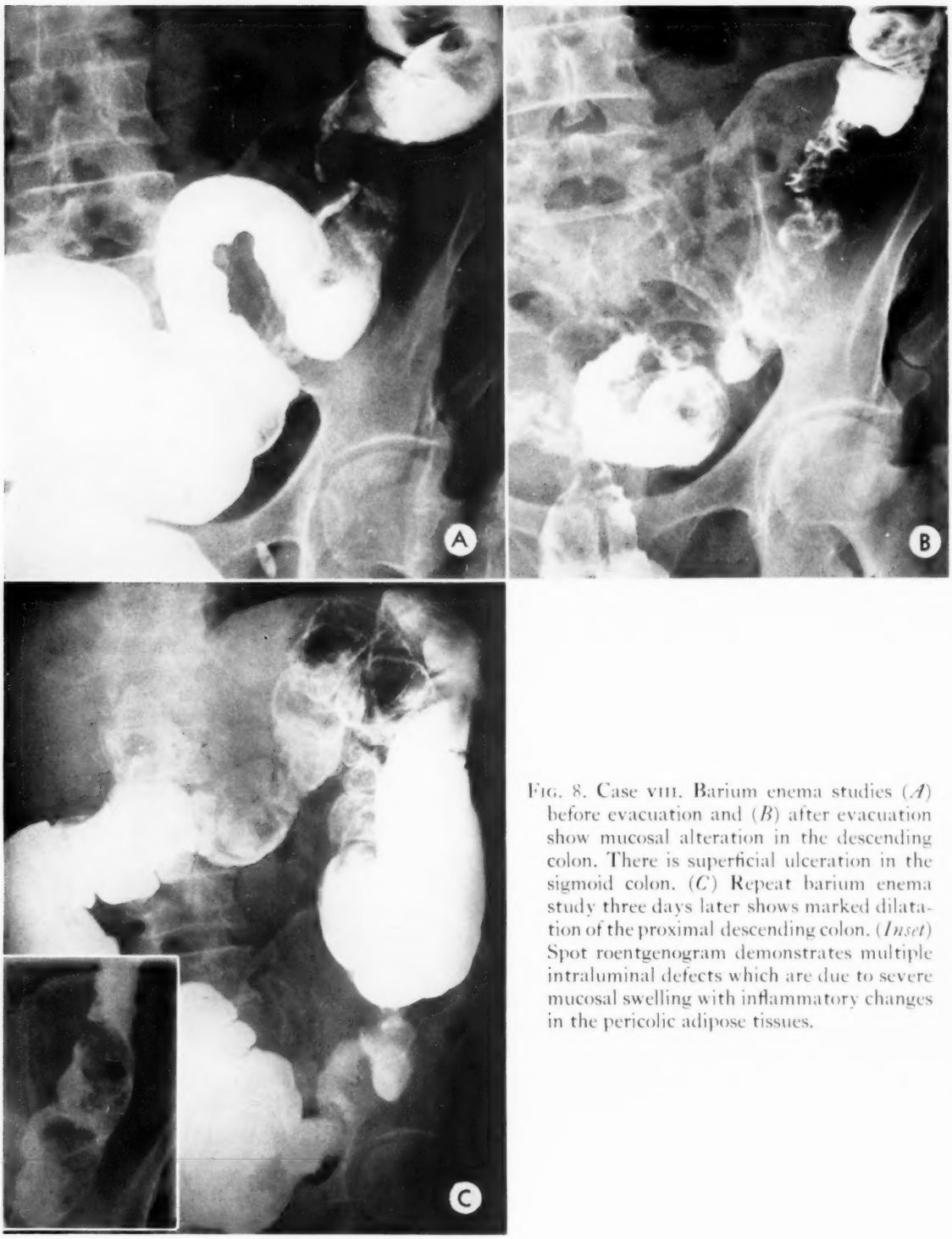

FIG. 8. Case VIII. Barium enema studies (*A*) before evacuation and (*B*) after evacuation show mucosal alteration in the descending colon. There is superficial ulceration in the sigmoid colon. (*C*) Repeat barium enema study three days later shows marked dilatation of the proximal descending colon. (*Inset*) Spot roentgenogram demonstrates multiple intraluminal defects which are due to severe mucosal swelling with inflammatory changes in the pericolic adipose tissues.

tion of a loop or loops of bowel with edema of intestinal walls and progressive increase of intraperitoneal fluid; (c) on contrast studies in the early cases, marked swelling of the mucosal folds with thickened bowel wall and, occasionally, an intraluminal pseudotumor formation.

2. In subacute and chronic cases, vague abdominal pain which may be anginal in nature. The plain roentgenograms may often reveal no abnormalities. Contrast substances, however, may outline narrowing of the bowel lumen with fixation and ulceration, simulating regional ileitis or ulcerative colitis, with or without evidence of malabsorption syndrome.

Any patient who develops "ulcerative colitis," "regional enteritis," or malabsorption syndrome after aortic grafting or cardiac surgical procedures should be suspected of mesenteric vascular disease until proved otherwise.

It is to be noted that aortography may or may not demonstrate occlusion or diminution of the mesenteric vascular flow. At our present state of knowledge, visualization of apparently patent mesenteric arteries should not exclude the diagnosis. It is quite possible that a change more subtle than macroscopically visible diminution of vascular flow in the larger arteries is to be dealt with. If the mesenteric artery is visualized, consideration should be directed to the possibility of mesenteric *venous* thrombosis. It is not illogical to believe that some of the more chronic enteric diseases of vascular origin may be due primarily to mesenteric vein rather than mesenteric artery disease; in certain instances both have been proved to be present.

CONCLUSIONS

It has been demonstrated that mesenteric vascular disease may exhibit a variety of patterns, both on plain roentgenograms and by contrast studies. Cases of localized tumefaction, malabsorption syndrome, "regional enteritis" and "ulcerative colitis" may have their origin from vascular occlusion or insufficiency.

Because the bowel lends itself so admirably to roentgenologic study, these possibilities should be kept in mind. In addition to plain roentgenograms and barium studies, aortography and mesenteric arteriography may aid in establishing the diagnosis, and further may be of valuable assistance in clinical management. The present day improvement in bowel and vascular surgery offers the possibility of altering an otherwise grave prognosis.[13]

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RETROGRADE ENTEROGRAPHY*

A NEW METHOD FOR THE ROENTGENOLOGIC STUDY OF THE SMALL BOWEL

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THE development of the roentgenologic study of the small intestine proceeded through the following evolution:

1. The standard gastrointestinal series. In this examination, done primarily for investigation of the upper gastrointestinal tract, a single delayed roentgenogram is taken at four or six hours, chiefly to determine whether any gastric retention is present. An incidental demonstration of a segment of the small bowel is obtained.

2. The "motility series." This consists of roentgenography every half hour from the time of ingestion of the barium suspension until the head of the barium column reaches the cecum, and is usually supplemented by spot roentgenograms of the ileocecal region.

3. The "small intestinal enema" study. This was devised by Schatzki[6] and is not an enema in the sense of the barium filling the small bowel from below upwards but consists of the passing of a Levine or Miller-Abbott tube into the duodenum, and injecting the barium suspension through it.

4. The "modified small bowel enema" study. As described by Blodgett[1] in this method a standard Miller-Abbott tube is used, first to decompress the distended bowel, then a thin barium suspension is introduced just proximal to the small bowel obstruction.

THE PRESENT METHOD

There are three main reasons for the present unsatisfactory state of roentgen examination of the small bowel: (a) the obscuration produced by the overlapping of barium-filled coils of bowel; (b) the effect of peristalsis and spasm, resulting in unfilled and partly-filled segments of bowel, as well as deep contractures; and (c) most physicians are not "small bowel minded" which often leads to unsuspected diagnoses being made only after death.

The method to be described, while not advocated for routine purposes, may prove to be helpful in selected cases. In principle, it follows that of the colonic barium enema study in which the barium fills the bowel from below upwards. In practice it consists of passing a 2-lumen intestinal tube, by which the small bowel is sealed off at a given point by a latex balloon, following which barium is introduced above the distended balloon and fills the bowel above it, each function being achieved through a separate lumen.* The present practice is to modify a standard Miller-Abbott tube by soldering its tip so that there is no communication of the larger lumen of the tube with the bowel after the terminal balloon has been inflated (Fig. 1), one lumen being used to distend the balloon and seal off the bowel, the other for the introduction of the barium suspension or air, or the washing out of the proximal part of the bowel with saline solution. A segment consisting of about one fourth of the small bowel, or less, may be examined fluoroscopically and

* The method was devised by the senior author thirteen years ago as the result of research in pernicious anemia and originally he designed four types of 2-, 3-, and 4-lumen intestinal tubes which were produced by the kindness of Geo. P. Pilling Co. of Philadelphia.

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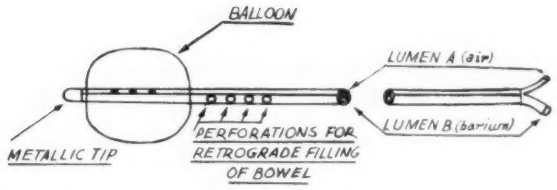

FIG. 1. *Diagram of Senior Author's (E.A.G.) Intestinal Tube (2-lumen model).* Lumen A is used to inflate or deflate the latex balloon at the tip of the tube. The inflated balloon acts as a seal or barrier in the small bowel. Lumen B carries the barium suspension, which is delivered through perforations in the rubber tube situated proximal to the inflated balloon.

roentgenographically, the filling of the bowel may be started or stopped at will, and the method permits double contrast or postevacuation roentgenograms to be made (Fig. 2, *A* and *B*).

During an examination at Bellevue Hospital of 16 patients without previously demonstrable small bowel lesions, roentgenograms of almost diagrammatic precision, resembling those seen of the large bowel after a barium enema, have been obtained. The passage of the tube into the large bowel presented some difficulty at the beginning, but once experience had been gained, not a single failure was encountered. The two difficulties at the beginning were gagging of the patient while the tube was being passed through the nasopharynx, and failure to obtain entry of the tube through the pylorus.

INTUBATION PROCEDURE

If a considerable number of cases are to be investigated, it is better, as pointed out to the senior author by Dr. Ross Golden,[4] that one person, preferably a nurse, be trained and entrusted to carry out the intubation procedure regularly. If there is doubt as to whether the pylorus has been entered, the patient should be taken to the radiologic department for fluoroscopy. As experience is gained by the operator, the procedure becomes less and less unpleasant to the patient. The gagging which patients (especially if they are apprehensive) may feel when the tube is passed through the nasopharynx might be overcome by introducing 3–5 cc. of metallic mercury into

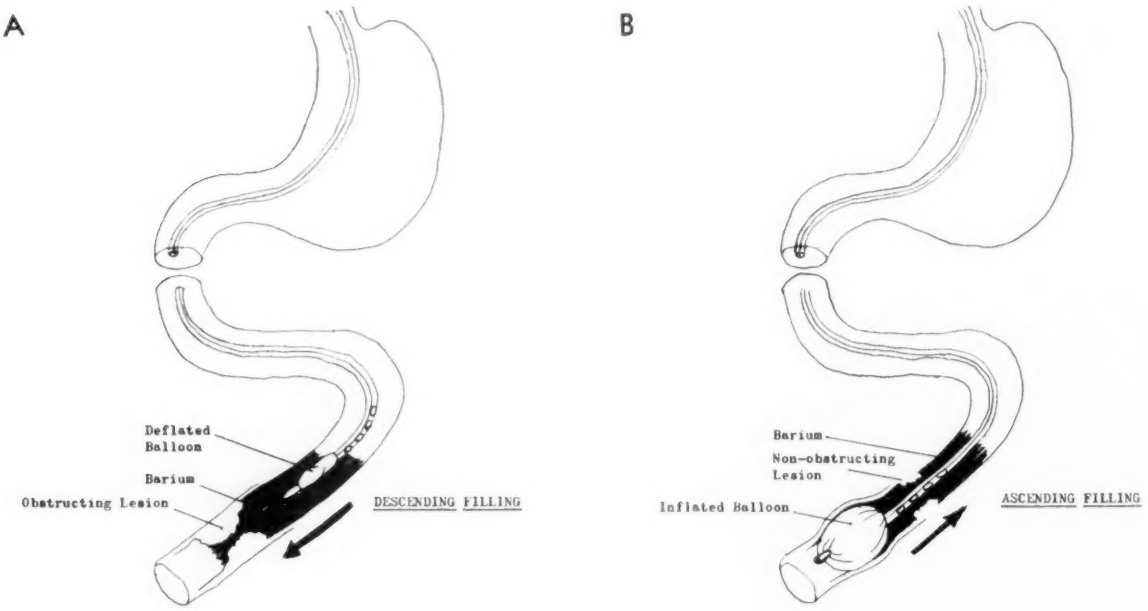

FIG. 2. (*A*) In the presence of obstructing lesions, the balloon is collapsed and barium runs forward to the site of obstruction. (*B*) In the presence of non-obstructing lesions, the balloon is passed beyond the site of the lesion, then inflated, and barium is introduced in a retrograde manner outlining the diseased segment.

the balloon *before* the tube is passed. Very nervous patients may be given a sedative or tranquilizer an hour or two before intubation. The procedure as performed by us was as follows:

1. The balloon was first inflated and examined in a good light for possible flaws and checked for leaks by submersion in water. The larger lumen of the intestinal tube was flushed out with water to ensure that there would be no obstruction to the flow of barium through it.

2. A 1 per cent pontocaine solution was sprayed into the larger and freer of the patient's nostrils. If both nasal cavities were too narrow to permit passage of the tube, the patient's tongue was depressed and the tube was passed orally.

3. The tube head, including the deflated balloon, and several feet of rubber tubing below it, were liberally lubricated with KY jelly. The patient was instructed to swallow water continually as the tube was carried down the esophagus.

4. With the tube head in the stomach, the patient was turned on his left side in order to facilitate the aspiration of the gastric contents.

5. The patient was then turned on his right side, in order that gravity might aid in directing the balloon from the fundus through the pylorus, and instructed to swallow ½ to 1 inch of the tube every fifteen or twenty minutes until the 75 mm. mark (the "111" mark) reached the nostril. The tube was then taped to the cheek.

6. Two tests were now made to make certain that the tip of the tube was actually in the duodenum. Aspiration through the large lumen was carried out, and if clear yellow bile was drawn into the syringe this indicated that the duodenum had been reached. The second check was to inflate the balloon moderately through the smaller lumen, with about 20 cc. of air. If there was resistance in the syringe, or backward movement of the piston, this indicated that the balloon lay within the relatively narrow confines of the duodenum. Routine fluoroscopy to make certain that the tip was in the duodenum became unnecessary as experience was gained and these two reliable signs were present.

With the mercury in the balloon, the tube passed quickly down the esophagus. The second obstacle, passage of the tube into the duodenum hardly seemed formidable after the preliminary use of mercury was begun. If, as happened only once, pylorospasm seemed to be the cause of delay at the pylorus, the patient was given amyl nitrite by inhalation. The use of the antispasmodic octin has been advocated for this purpose, as well as the injection of morphine and atropine before intubation by Cantor.[3] We have occasionally injected 1/6 gr. morphine or 50 mg. demerol hydrochloride before intubating nervous, apprehensive patients.

When the balloon was situated in the third part of the duodenum, it was partially inflated with 20 cc. of air so that it could act as a bolus and be propelled by the peristaltic activity of the bowel. The patient was permitted to push the tube gently down but at a speed no greater than one or two feet per hour.

In a series of 800 patients, Golden has reported 90 per cent of successful intubations.

ROENTGENOGRAPHIC PROCEDURE

The following roentgenographic procedure was temporarily adopted in order to assess the type of visualization of the small bowel that is obtained by the retrograde method. Experience has suggested further improvements which will be discussed below.

1. When the tube was in place in the lower small bowel, as determined by a scout roentgenogram, the balloon was inflated with 50 to 60 cc. of air, in order to seal off the small bowel.

2. About 20 cc. of barium suspension was instilled through the large lumen of the tube and a 14×17 inch roentgenogram (Bucky) was made. This and all subsequent roentgenograms were immediately processed before additional barium was instilled.

3. Additional barium suspension in increments of about 200 cc. was introduced until there was an indication of beginning overlapping of barium-filled coils of bowel, or until the patient became uncomfortable. The discomfort is usually a mild abdominal cramp, due mainly to the pressure of the inflated balloon and to some extent probably to the weight of the barium column resting upon it. It was usually found that 600 cc. of barium suspension could be tolerated.

Apparently three factors may influence the amount of barium that can be tolerated without too much discomfort: (a) the density of the barium suspension, (b) the temperature of the suspension, and (c) the speed of instillation. Suspensions of 1:4 and 1:8 seemed best. Cold supension was less well tolerated than warm (about 37°C. seemed best). The suspension must not be instilled too rapidly, else cramps are provoked before an adequate amount can be given.

POSSIBLE IMPROVEMENTS IN ROENTGENOGRAPHIC PROCEDURE

It is obvious that the addition of fluoroscopy greatly enhances the value of the technique because one may take advantage of the complete filling which is provided by the retrograde method as by no other. Thus, for example, if a suspicious section is encountered while the slow filling of the small bowel is being observed fluoroscopically, the operator may instantly halt filling and proceed to take spot roentgenograms, with or without compression, double contrast roentgenograms, postevacuation roentgenograms or ordinary Bucky roentgenograms. The filling may then be resumed at the operator's will. If desired he might change to a barium suspension of a different density. Everything should be in its proper place in the darkened room and easily accessible.

The use of a water manometer in order to produce a standard optimum air pressure within the inflated balloon might be an improvement, since this pressure would cause least obstruction and discomfort while also acting as an effective seal. The delivery of the barium suspension by gravity rather than by a 50 cc. Luer syringe, as was done in this study, might be a further improvement.

ILLUSTRATIVE CASES

The 16 patients selected for this study were from the surgical and medical wards of Bellevue Hospital, and the majority were not suspected of having any small bowel pathology. The purpose was to demonstrate the small intestine and, as will be seen, the altogether superior type of visualization that is obtained by retrograde enterography. The following 5 cases are presented as examples.

Case 1. E.G., an eighty-one year old male, was admitted to the hospital with a clinical diagnosis of intestinal obstruction secondary to adhesions. The patient had had several previous admissions for the same condition, which always cleared after decompression with an intestinal intubation. During the present admission, four days after decompression had eliminated all clinical and roentgenographic signs of ileus, retrograde enterography was undertaken. Figure 3*A* shows the tip of the special intestinal tube in the mid-jejunum and the inflated balloon is represented as a radiolucent, elliptical shadow at the tip. That the inflated balloon has completely sealed off the bowel is evident from the fact that the barium column lies entirely proximal to the balloon. The filling of the bowel was produced by the instillation of 75 cc. of 1:4 barium suspension by volume. Figure 3*B* shows a larger section of bowel which was filled by a total of 250 cc. of barium suspension. The excellent detail is striking. The Kerkring folds are prominent and well separated. The appearance has somewhat the quality of an anatomic drawing, which has not been obtained by any other method.

The bowel in this case is of wider diameter than is usual, apparently due to stretching and dilatation behind the distended balloon. Figure 3*C* shows how markedly reduced in diameter the bowel became after the balloon was deflated. This brought about the rapid filling in a descending direction of the bowel distally to the

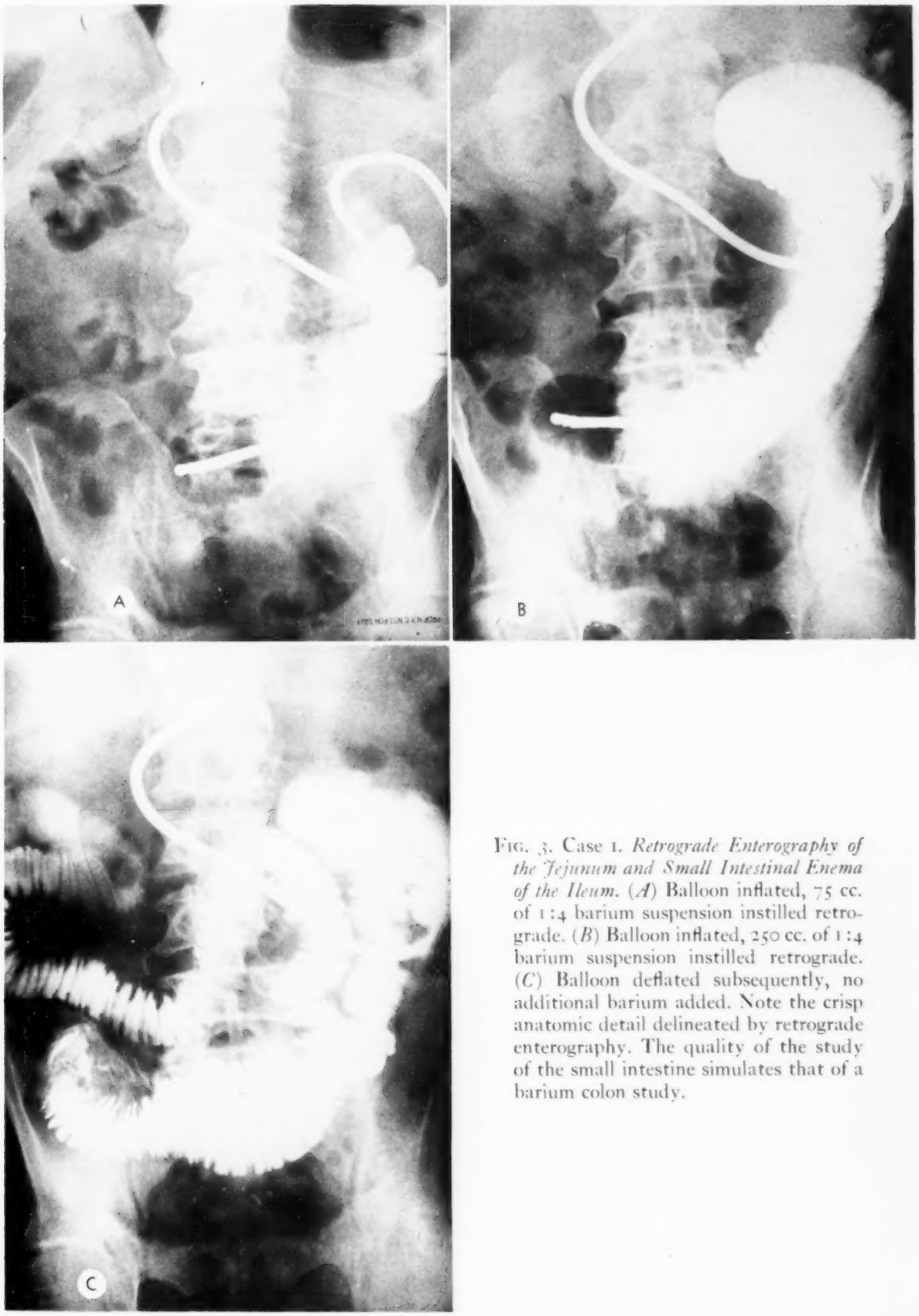

FIG. 3. Case 1. *Retrograde Enterography of the Jejunum and Small Intestinal Enema of the Ileum.* (*A*) Balloon inflated, 75 cc. of 1:4 barium suspension instilled retrograde. (*B*) Balloon inflated, 250 cc. of 1:4 barium suspension instilled retrograde. (*C*) Balloon deflated subsequently, no additional barium added. Note the crisp anatomic detail delineated by retrograde enterography. The quality of the study of the small intestine simulates that of a barium colon study.

Fig. 4. Case II. *Retrograde Enterography of the Ileum.* Balloon inflated, 200 cc. of 1:4 barium suspension instilled retrograde. Note the crisp anatomic detail of the ileum. Note the identification of mucosal folds, as compared with the usual clumping and puddling obtained by the conventional barium meal study.

balloon. The marked reduction in the diameter of the bowel suggests an atonic condition.

The descending filling of the lower small intestine which occurred upon deflation of the balloon is in effect an "intestinal enema" (Schatzki[6]) with the "enema" starting in the mid-jejunum rather than in the duodenum. These roentgenograms suggest that it is possible to examine the entire small bowel by the retrograde method by first taking ascending roentgenograms, then deflating the balloon and thereby getting descending filling for roentgenograms of the rest of the small bowel.

Case II. J.S., a fifty-two year old white Polish male was an extremely sensitive gagger and was given an injection of 50 mg. of demerol both before intubation and five hours later, when he complained of cramps during roentgenography. This was immediately followed by vomiting which curtailed the examination. Figure 4, obtained after instillation of 200 cc. of barium suspension, shows the tip of the modified Miller-Abbott tube in the lower ileum. The balloon contains both air and mercury. The diameter of the barium-filled ileum is only slightly larger than that seen after oral administration of the standard motor meal, but the column of the barium-filled bowel is continuous and the details are strikingly clear, in contrast to the usual puddling and clumping ordinarily seen in the lower ileum. There is considerable looping and curling of the bowel around the intestinal tube, apparently due to the contraction of the longitudinal muscles

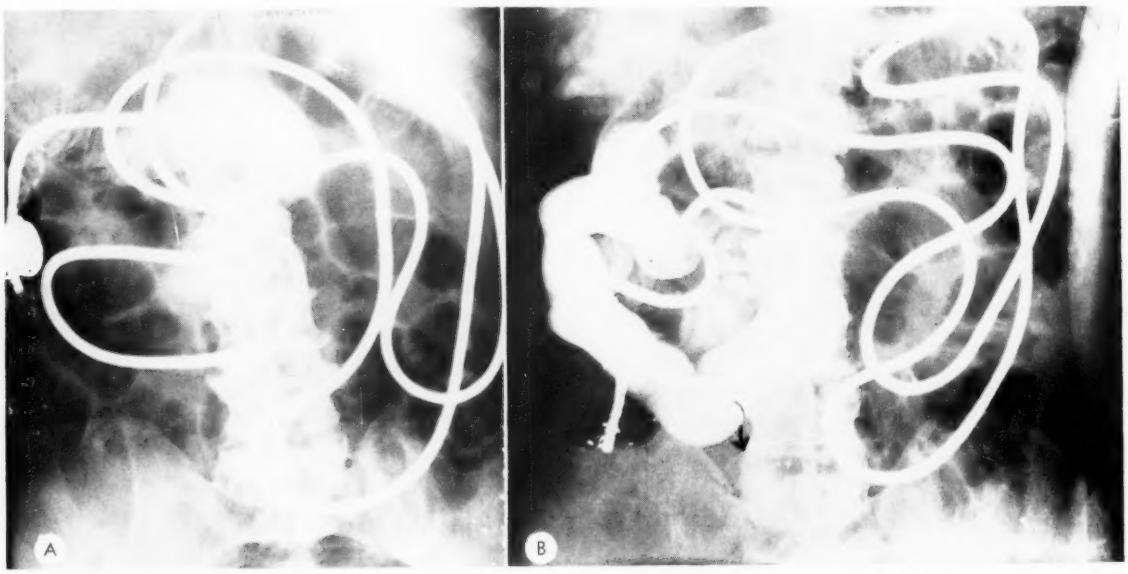

Fig. 5. Case III. *Use of Extremely Thin Barium Mixture in Retrograde Enterography.* (*A*) Balloon inflated, 200 cc. of thin barium suspension instilled retrograde. This was too thin a mixture. (*B*) Additional 200 cc. of 1:8 barium suspension was instilled. Note the filmy "double contrast effect" achieved with the use of dilute barium.

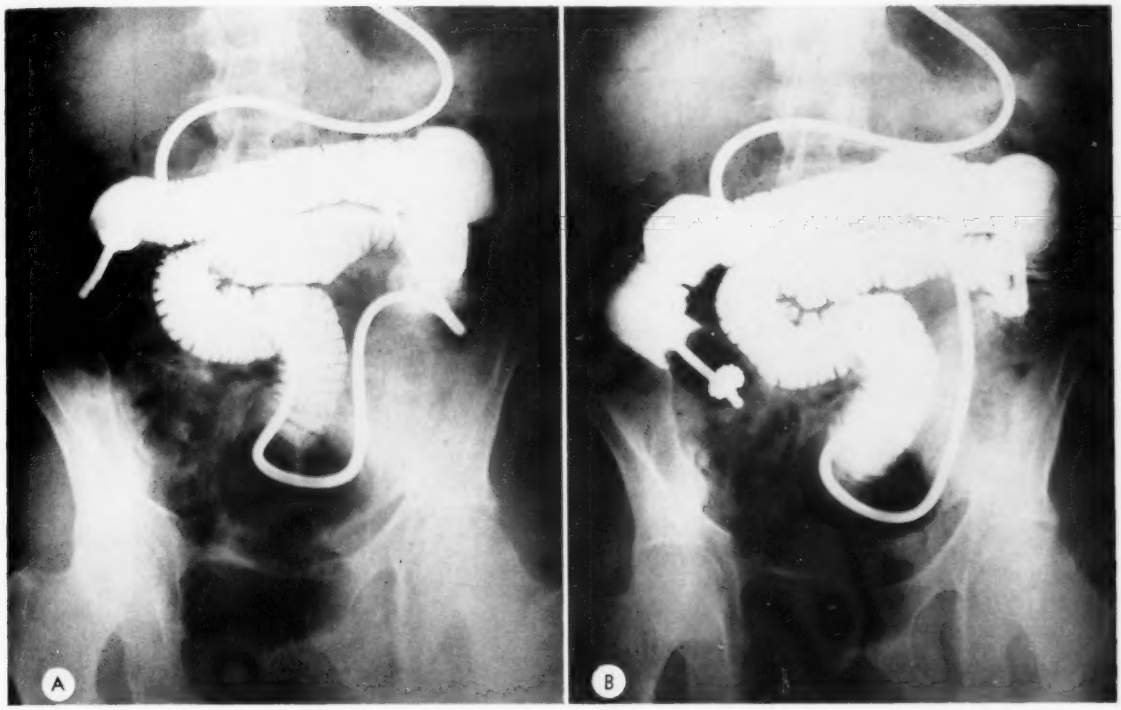

Fig. 6. Case iv. *Use of Very Thick Barium in Retrograde Enterography.* (*A* and *B*) Instillation of 200 cc. and 400 cc. of 1:4 barium suspension respectively. Note the extreme radiopacity of the barium filled intestine, and the significant distention. This study approaches the characteristics of a true barium enema examination with each of the folds neatly dilated. It, however, obscures the definition of the fine mucosal pattern.

producing marked shortening of the bowel. This appearance is indicative of a state of hypertonicity.

Case iii. N.S., a forty-five year old white male, was admitted to the hospital for investigation of vague abdominal pain. Figure 5*A* shows a segment of the ileum visualized after the instillation of 200 cc. of barium suspension. This apparently is too thin a mixture because short sections of bowel are seen to be unfilled. Figure 5*B* shows the result of instillation of an additional 200 cc. of a somewhat denser barium suspension (1:8 by volume). In the right lower quadrant (see arrow) the loops of small intestine show a pattern similar to that seen on air contrast studies of the colon. It may be that by the use of dilute barium suspensions mucosal relief patterns can be obtained.

Case iv. W.H., a twenty-four year old Puerto Rican male, was admitted to the hospital because of continuing abdominal pain and melena following sphincterectomy for pancreatitis on a previous admission. Figure 6, *A* and *B* was made after instillation of 200 cc. and 400 cc. of barium, respectively. As the barium is increased in amount it is noted, first, that there is loss of detail as a result of excessive radiopacity, and secondly, that the loops of small intestine become somewhat more distended and widened and are pushed more closely together, making a study of the borders more difficult. This points up the necessity of using thinner mixtures, whereas Case iii indicated the unsuitability of mixtures that are too thin. Nevertheless, these several loops of small bowel have somewhat the appearance observed on roentgenograms taken after colonic barium enema administration.

Case v. A.V., a forty-eight year old white Italian male, was admitted to Bellevue Hospital for gastroenterologic investigation. In this patient air-contrast studies of the small bowel were attempted with the retrograde method. Figure 7 shows the roentgenogram obtained after the instillation of 200 cc. of air and 50 cc. of barium suspension. The segment of ileum immediately proximal to the inflated balloon shows double contrast visualization at some distance proximal to the balloon seal.

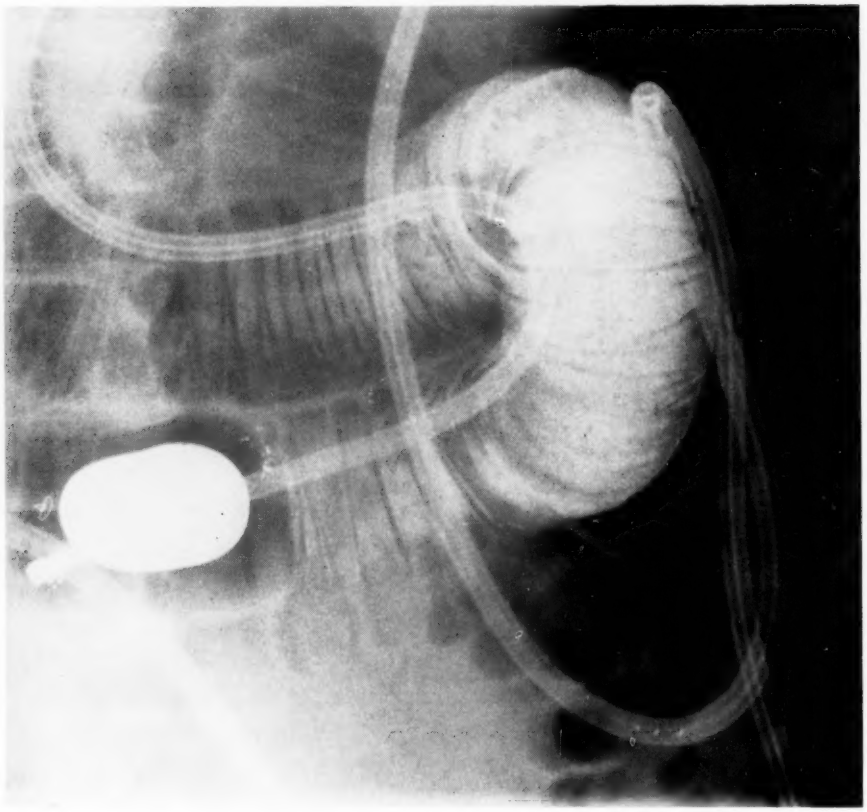

FIG. 7. Case V. *Air Contrast Study with Retrograde Enterography.* Instillation of 200 cc. of air and 50 cc. of barium 1:4 suspension. A true "air contrast" study of a portion of the small intestine is secured.

ADVANTAGES OF RETROGRADE ENTEROGRAPHY

1. *Superior Visualization.* By methods of oral administration of barium suspension, the small bowel exhibits roentgenographically a feathery mucosal pattern or, in those portions that are better filled, it gives the picture of sausage-shaped masses separated by unfilled or poorly-filled sections of bowel, or of clumped or puddled masses. By the retrograde method, roentgenograms are produced which show the bowel as a continuous, uninterrupted column of normal width or frequently wider than normal and often with diagrammatic precision. The detail is much greater and spastic contractures that might ordinarily serve to conceal or confuse the presence of an organic lesion are ironed out because the barium column is firmly fixed above the inflated balloon and filling proceeds from that point in a direction opposite to that of peristalsis.

2. *Complete Control of Filling.* By retrograde enterography the operator can, at will, either completely stop the filling, start it again, or change the speed of filling. Even by the intestinal "enema" of Schatzki, though the flow of barium through the duodenal tube may be clamped off, peristalsis continues to propel the barium rapidly through the small bowel, and so, once filling has commenced, it cannot be stopped or even slowed down. By our method filling is under complete control of the operator.

The speed of filling is also under complete control, depending upon the amount and temperature and the rapidity of instillation of the barium supension. When the instillation is stopped the picture is immediately frozen *in status quo* until filling is resumed. At this stage spot roentgenograms can be taken, air contrast studies made, or postevacuation roentgenograms taken.

Plain or Bucky roentgenograms may be made instantly.

3. *Choice of Techniques.* Whereas some other motility series may require as many as sixteen roentgenographic sessions, one session using retrograde enterography may be sufficient for the examination of the whole small bowel. With the tip of the tube in the upper jejunum, the jejunum and the duodenum may be filled slowly by the ascending method and then, by slow deflation of the balloon the rest of the small bowel may be filled by the descending or "enema" technique of Schatzki from the jejunum. However, if a lesion is suspected in a certain region of the small bowel and if the tip of the tube is introduced below this point, the affected area may be covered by the ascending technique. In both instances, the retrograde method permits spot roentgenography, double contrast roentgenography and postevacuation roentgenography, as well as fluoroscopic examination, as shown from the foregoing.

DISADVANTAGE OF THE RETROGRADE TECHNIQUE

The accompanying discomfort of intestinal intubation can be greatly minimized as experience is gained by the operator. The time consumed by this technique can be actually less than by other improved methods of the barium series.

INDICATIONS AND USES OF THE RETROGRADE TECHNIQUE

When the senior author first consulted Dr. Ross Golden in this matter he gave the following list of probable indications:

1. In unexplained intestinal bleeding which may be due to Meckel's diverticulum or other causes.
2. In unexplained diarrhea.
3. In unexplained abdominal pain, especially when periumbilical.
4. When routine barium studies have not revealed the diagnosis, and when perhaps operation is being considered—if the patient's condition is not too urgent.

A further important indication is where ordinary barium studies lead the radiologist to suspect a segmental small bowel lesion, such as an early neoplasm, inflammatory disease, regional ileitis, Meckel's diverticulum, adhesions, diverticula or small bowel obstruction. In this situation, an opportunity exists for a leisurely detailed controlled study of the area under suspicion.

NEED FOR AN IMPROVED METHOD

Botsford and Seibel,[2] in a study of benign neoplasms of the small bowel, conclude that it is exceedingly rare for a benign tumor to be discovered *per se*. Some produce obstruction and ileus. Others are discovered because an intussusception is formed, and it is this complication that leads to recognition of the growth. The authors make the following plea for early diagnosis:

"The small intestine comprises the longest portion of the alimentary canal, and yet receives small consideration in the diagnosis of intestinal tumors. The reason is the rarity of neoplasms of the small bowel. Such easy setting aside is in line with the attitude toward these tumors in general and can no longer be tolerated by the profession. Moreover, tumors of the small intestine are possibly curable in the early stages and certainly the small bowel is heir to a large variety of benign tumors that may cause symptoms. The diagnosis is usually obscure and unless the physician entertains the possibility they will remain obscure."

Golden points out that there is also difficulty in differentiating malignant tumors of the small bowel from inflammatory conditions. He feels that: "The character of the inner surfaces of the two areas in the two conditions . . . will not permit a differentiation."

Golden[3] also states that malignant tumors of the small bowel, like benign tumors, are not diagnosed roentgenographically until late in the disease when the tumor has produced obstruction and ileus. Thus, Golden notes that a patient with

polypoid carcinoma had "such marked segmentation of the barium-filled loops that the filling defect was not demonstrated."

SUMMARY

Retrograde enterography, a new method for roentgenologic examination of the small bowel, is described and the indications for its use are given.

Walter Lentino, M.D.
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ADDENDUM

The senior author has had the opportunity of examining by retrograde enterography a few cases with small bowel pathology which he hopes to incorporate in a future report.

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PROLONGED CATHARTIC ABUSE RESULTING IN ROENTGEN EVIDENCE SUGGESTIVE OF ENTEROCOLITIS

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THERE are only a few indications for the use of a cathartic and, contrary to popular belief, chronic constipation is not one of them. Many people believe that a daily bowel movement is necessary for good health, and, if one is missed, the patient resorts to use of a cathartic to promote bowel evacuation, instead of establishing habits that favor regular elimination, such as eating enough foods containing indigestible bulk, drinking plenty of fluids, establishing a regular time for bowel movements, and proper physical activity. As a result of thorough catharsis, several days may elapse before a normal bowel movement can occur. Therefore, the patient continues to use the cathartic because he believes his bowels will not move without it.

Many of the proprietary cathartic preparations contain one or more of the cathartics the action of which depends on irritation of the intestinal mucosa and increased motor activity of the bowel. Therefore, habitual use of these drugs may cause the bowel to become so spastic that it will not move without them, and thus a vicious circle is set up. To many a patient, the use of a cathartic has become as routine as eating and sleeping, and he often neglects to mention it to the physician unless he is questioned directly about it.

The active ingredients in each tablet of one of the proprietary preparations that was used by many of the patients whom we will describe are as follows: aloin $\frac{1}{4}$ grain, podophyllum $\frac{1}{6}$ grain, extract of cascara $\frac{1}{4}$ grain, extract of belladonna $\frac{1}{8}$ grain, and strychnine sulfate $\frac{1}{60}$ grain.

Members of the irritant group of cathartics produce changes in the motor activity of the bowel that are of such a nature that an abnormal roentgenographic appearance of the colon and terminal ileum may result.

ROENTGENOLOGIC FINDINGS

Other authors[1–3] have described the roentgenologic abnormalities in the colon and terminal ileum associated with prolonged use of cathartics in 11 patients.

In the nine year period 1947 through 1955, a total of 265 patients at the Mayo Clinic gave clinical histories that the internists interpreted as being consistent with chronic cathartic abuse. Most of those who underwent roentgenographic examination of the colon were reported by the radiologist as having negative findings. Others were reported as having dolichocolon, large redundant colon, atonic colon, large atonic colon, or large-caliber colon; the roentgenograms had been discarded, however, and so were not available for review. Others were said to have "burned out" enterocolitis and some were recognized, mostly by one of us (H.M.W.), as having a condition due to chronic cathartic abuse.

Because of the confusion that may result in the differential diagnosis of colonic changes apparently due to cathartic abuse and those of relatively inactive chronic ulcerative colitis, it was deemed worth while to report our roentgenologic findings in these patients. We were able to collect 27 cases in which there was a history of chronic cathartic usage, usually in excessive daily dosage, and in which roentgenologic findings in the colon were abnormal.

In the roentgenograms of these patients, the most striking finding was complete or

* Deceased.
† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

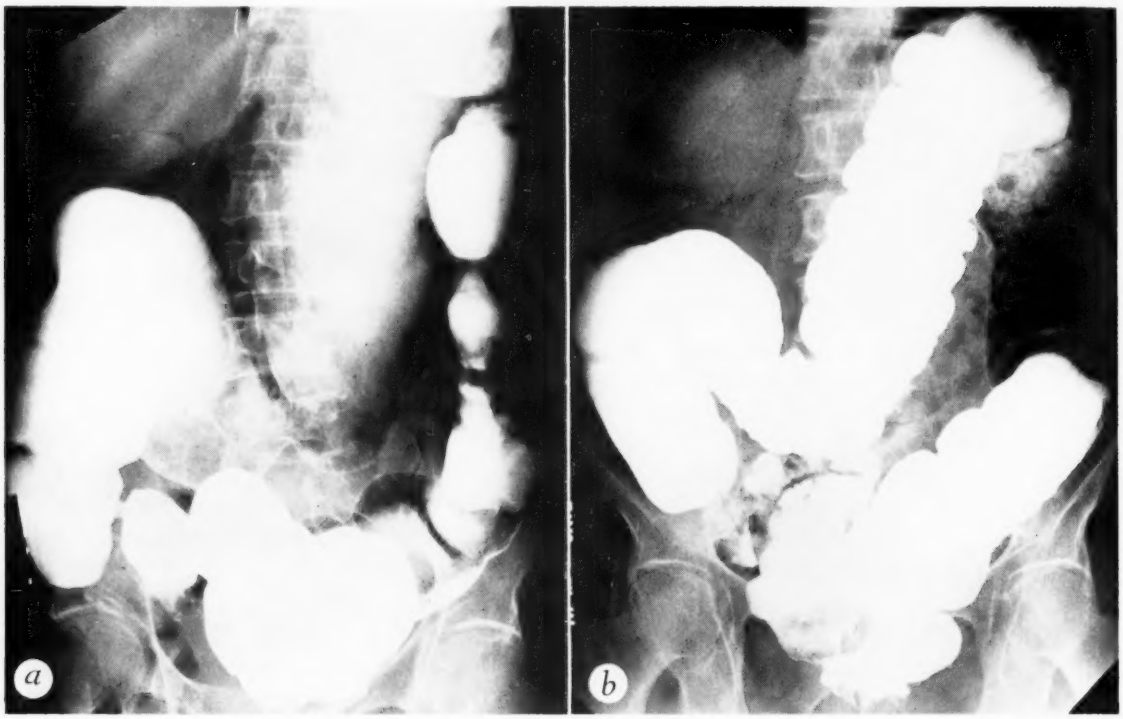

FIG. 1. (*a*) Postevacuation roentgenogram showing constricted segment in transverse colon (see text). (*b*) Repeat examination six days later reveals no evidence of constricted segment.

nearly complete absence of the normal haustral markings of the colon. In long-standing cases, the entire colon as well as the terminal ileum was involved. In other cases, only the right side of the colon and the terminal ileum were affected. The outline of the mucosa as seen in relief was entirely smooth in the portions of the bowel affected, and there was never the irregularity or evidence of ulceration that is seen in chronic ulcerative colitis. There was little or no evidence of thickening or stiffening of the bowel such as that which occurs in chronic ulcerative colitis. On the contrary, the bowel usually was very distensible, often dilating to several times the normal caliber without the patient complaining of urgent need for evacuation (Fig. 1*a*).

FIG. 2. Complete absence of haustral markings with smooth mucous membrane as seen in relief, shortening of right side of colon, and constricted segments in transverse colon.

Both on the roentgenograms and at fluoroscopic examination, one often sees segments of smooth constriction of the bowel lumen that appear to impede the retrograde flow of barium temporarily when observed fluoroscopically (Fig. 2). If these narrowed segments are observed fluoroscopically for several seconds or a few minutes, they are seen to dilate up to the caliber of the adjacent lumen, and other

segments that were originally filled may be observed to constrict. Thus, at the same time, there may be spastic constricted segments and apparently dilated atonic segments, suggesting a disordered neuromuscular function of the bowel wall.

The constricted segments have smooth, tapering extremities and the lumen tapers gradually, rather than abruptly as is seen with neoplastic involvement. These narrowed segments might be interpreted as strictures of the bowel lumen if they were not observed over an extended interval at fluoroscopic examination or if they appeared on several roentgenograms taken in quick succession before the constricted segment had an opportunity to dilate.

In this series, the mucosal pattern in the constricted segments was always very smooth, and without any evidence of distortion such as that caused by neoplastic or inflammatory involvement. In several cases the cecum, ascending colon and hepatic flexure appeared to be shortened (Fig. 2, 3, 4, *a* and *b*, and 5, *a* and *b*). However, at fluoroscopic examination the segments would dilate up to a large caliber; hence, the changes were not fixed as in long-standing chronic ulcerative colitis but probably resulted from spasticity due to irritation by the cathartic.

The terminal ileum was dilated in many cases and showed absence of normal mucosal relief.

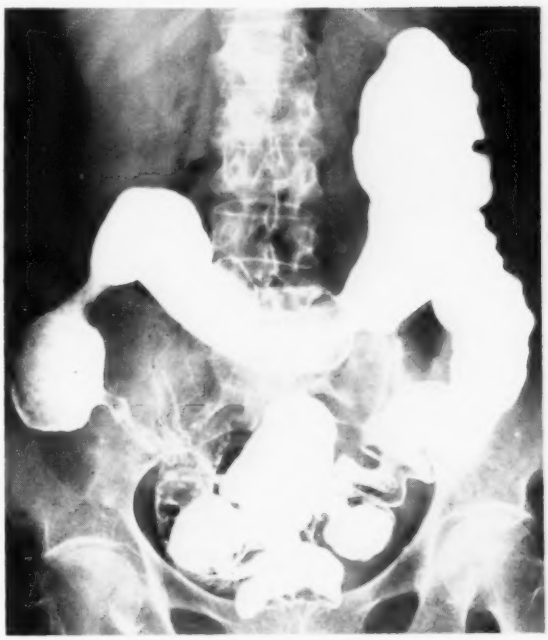

Fig. 3. Note absence of haustrations in colon proximal to splenic flexure, shortening of right side of colon, and constricted segment in ascending colon.

ILLUSTRATIVE CASES

Case I. A sixty-eight year old woman complained of pains in the stomach for years. She described these as "cramps all over all the time —day and night." She had always been constipated and had used a proprietary laxative at intervals ranging from two to three times weekly to daily for several years and seldom had a bowel movement without using a laxative. She had never had diarrhea or liquid stools. She took an enema about once a week.

Physical examination revealed diffuse tenderness along the course of the colon and especially in the right lower quadrant. Proctoscopic findings were negative to a distance of 24 cm., except for anal scarring with a subcutaneous sinus.

Roentgen examination of the colon with the aid of a barium enema revealed absence of haustral markings in the transverse and ascending colon and cecum, with shortening of the right side of the colon and a constricted segment in the ascending colon (Fig. 3).

Case II. A forty-six year old housewife gave a history of having taken two pills of a proprietary laxative (see contents listed on a previous page) daily for fifteen years. If she omitted the laxative, she complained of constipation. The laxative produced 5 to 15 liquid stools a day and the patient often noted undigested food in the stools, but never any blood or mucus.

Physical examination was noncontributory. Proctoscopic examination revealed anal contraction and anal fissure, but the mucous membrane was normal to 21 cm. The basal metabolic rate was −35 and −18 per cent on two occasions.

Roentgen examination with the aid of a barium enema revealed complete absence of haustral markings, with smooth mucous membrane as seen in relief. There were shortening of the right side of the colon and several con-

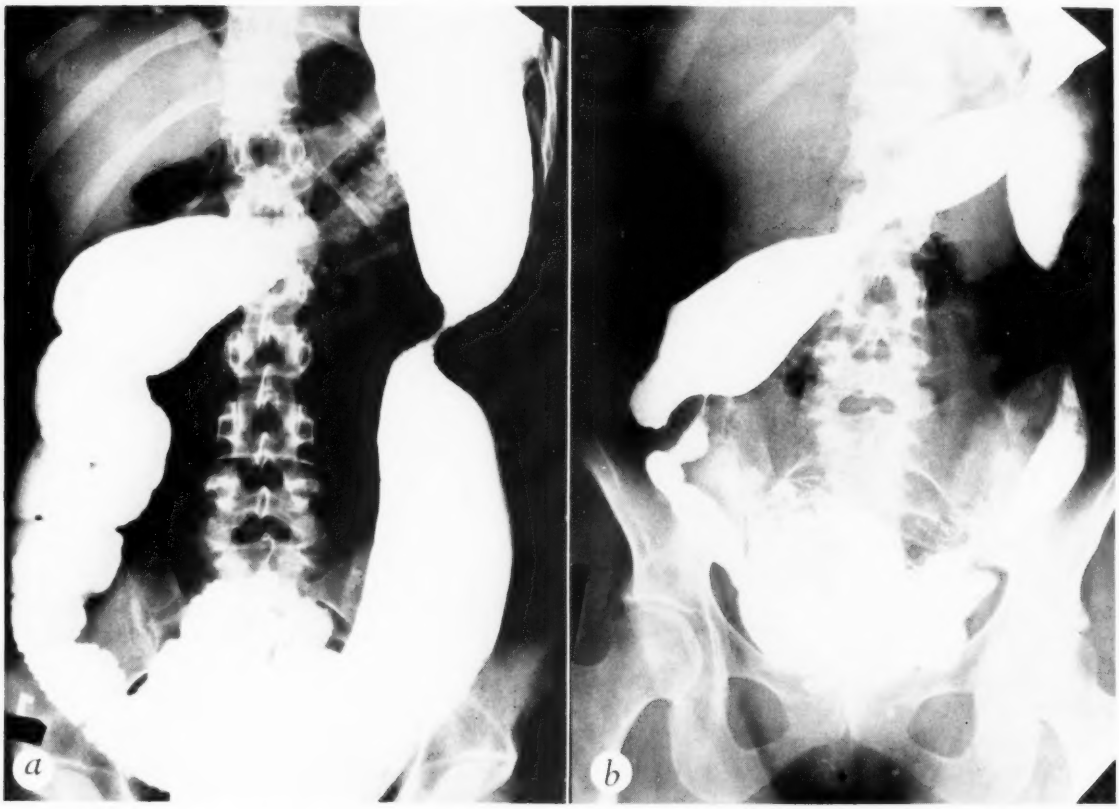

Fig. 4. (*a*) Colon filled with barium. (*b*) Postevacuation appearance. Note distensiblity of descending colon, and change in length of constricted segment in descending colon after evacuation. Also note complete absence of haustrations, smooth mucous membrane relief in entire colon and terminal ileum, and shortening of cecum and ascending colon.

stricted segments in the transverse colon (Fig. 2).

Case III. A thirty-seven year old unmarried woman complained of lassitude and fatigue of ten years' duration and gave a history of constant use of the proprietary cathartic the ingredients of which were listed previously. Physical examination and laboratory studies were noncontributory. Barium enema examination revealed complete absence of haustral markings in the entire colon. The mucous membrane as seen in relief was smooth in the entire colon and the terminal ileum. There was shortening of the cecum and ascending colon and there were constricted segments in the transverse and descending portions of the colon (Fig. 4, *a* and *b*).

Case IV. A fifty-year old white woman complained of pain and swelling of the abdomen of six months' duration. She also had had chronic constipation for twenty-three years, with almost daily use of cathartics and frequent enemas. In 1956, cholecystectomy was performed elsewhere and the surgeon described a distended colon, that appeared to be filled with fluid. He also described adhesions about the sigmoid colon, which he freed. Since then, the patient had continued to complain of low-grade generalized abdominal pain, bloating, and gurgling in the abdomen. Barium enema examination of the colon done prior to the patient's visit to the Mayo Clinic raised the question of a constricting lesion in the transverse colon.

Proctoscopic examination revealed anal fissure, contracture and hemorrhoids. The mucous membrane was normal for a distance of 24 cm. Barium enema examination revealed decreased number of haustral markings and smooth mucous membrane as viewed in relief in the colon (Fig. 1*a*). The terminal ileum was dilated, with smooth mucosal relief. There was a constricted segment in the transverse colon

overlying the lower part of the lumbar spinal column. This segment was observed for several minutes fluoroscopically, and it apparently persisted for several more minutes during evacuation of the enema and until after the postevacuation roentgenograms had been taken. Because the patient had been referred to the Mayo Clinic with suspicion of a lesion in the transverse colon, a repeat examination (Fig. 1*b*) was done six days later; the constriction was no longer present.

Case v. A fifty-six year old white woman had complained of persistent pain in the left flank and midback for fifteen years. She had noted fresh blood on defecation daily for one month. She had been constipated for years and used an irritant cathartic and milk of magnesia daily. Because of the blood in her stools and a strong family history of carcinoma of the colon, she had undergone barium enema examination elsewhere three weeks prior to being seen here, which had reportedly given negative results.

Proctoscopic examination here revealed anal fissure, contracture and hemorrhoids. The mucous membrane was normal to 24 cm. Barium enema examination revealed the changes of chronic cathartic abuse (Fig. 6*a*). The patient was instructed to stop using irritant cathartics and to use no laxative, if possible. Psyllium hydrophilic mucilloid (metamucil) was recommended for use if a laxative became necessary. Eleven months later, the patient returned and reported no change in her constipation. She had stopped using the irritant cathartics and had been taking 2 tablets of milk of magnesia every second night. She stated that she could not take metamucil. She also drank plenty of fruit juices, especially prune juice, and water. Barium enema examination of the colon (Fig. 6, *b* and *c*) revealed no significant change in the appearance of the colon since the previous examination.

Case vi. A thirty-eight year old married white nurse complained of low backache of four years' duration. She had had a right segmental oophorectomy for endometriosis four years prior to examination here. She also gave a history of chronic constipation with small stools and daily use of a proprietary cathartic. Barium enema examination of the colon revealed complete absence of haustral markings, smooth mucosal relief of the colon and terminal ileum, and apparent shortening of the cecum, ascending colon and transverse colon (Fig. 5, *a* and *b*).

Comment

In many of these cases, the disorder was diagnosed by the radiologist as "burned out" ulcerative colitis. However, it is apparent from the clinical histories of chronic constipation, constant long-term use of laxatives, absence of diarrhea with blood or mucus, absence of the frequent complications of chronic ulcerative colitis, and normal proctoscopic findings that these patients did not have idiopathic chronic ulcerative colitis. They did not have the marked shortening of the bowel, narrowing of the lumen, thickening and stiffening of the bowel wall, and ragged mucosal pattern that are caused by long-standing ulcerative colitis.

As pointed out by Heilbrun and Bernstein,[2] when the changes are limited to the right side of the colon, the roentgen appearance might be confused with that of amebic colitis; but the clinical findings of this disease are absent. Also, in most of our cases with involvement of the right side of the colon, the terminal ileum also was affected. Involvement of the terminal ileum is said not to occur in amebic infection. Right-sided ulcerative colitis with involvement of the terminal ileum might be closely simulated roentgenographically, but differentiation on clinical grounds should be easy.

The changes that we have described have been found only after use of the irritant cathartics, which irritate the intestinal mucosa and also increase motor activity of the bowel wall. This group of cathartics includes aloin, podophyllum, senna, cascara sagrada, phenolphthalein, castor oil, jalap, colocynth and elaterin. The changes described have not been seen following prolonged usage of the bulk and emollient cathartics.

Not only has usage of cathartic preparations been prolonged, but daily dosage in many instances has been much in excess of the recommended effective cathartic dose for a normal patient. One patient who was

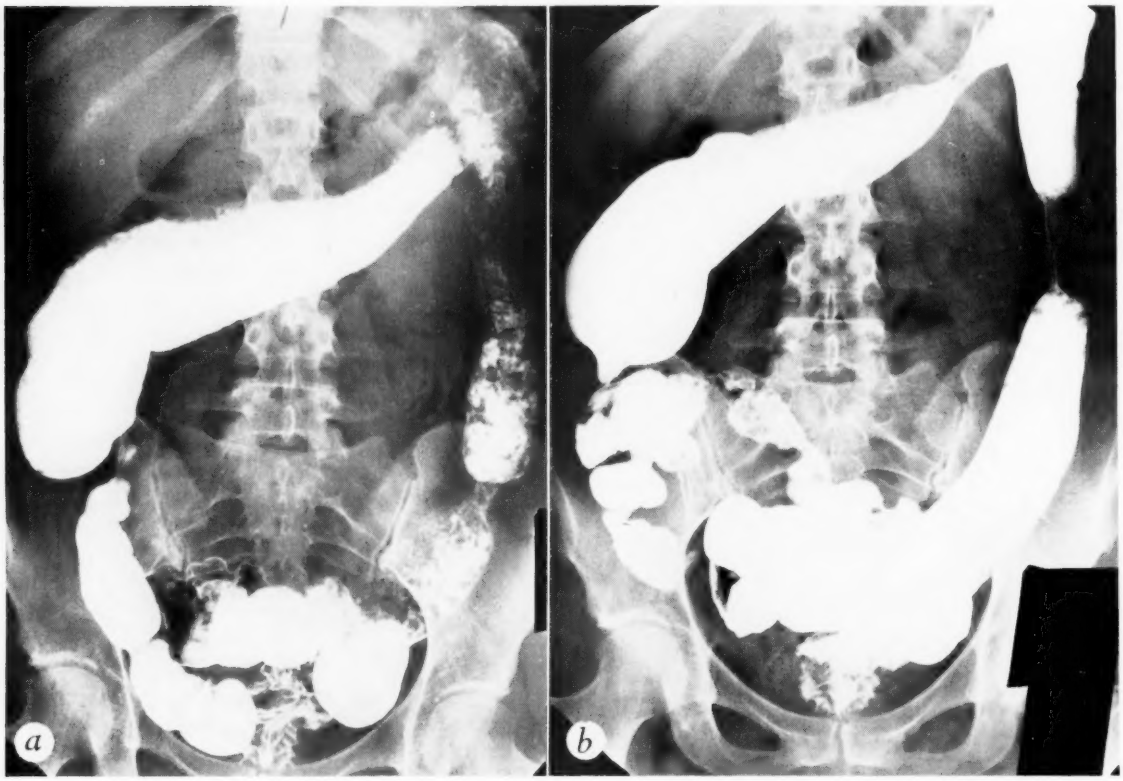

FIG. 5. (*a*) Postevacuation roentgenogram of colon reveals complete absence of haustral markings, smooth mucous membrane pattern, and apparent shortening of cecum, ascending colon and transverse colon. There are dilatation and smooth mucosal relief of terminal ileum. (*b*) Postevacuation roentgenogram after another examination made three days later. Constricted segments are present on both roentgenograms, but sites of constriction have changed.

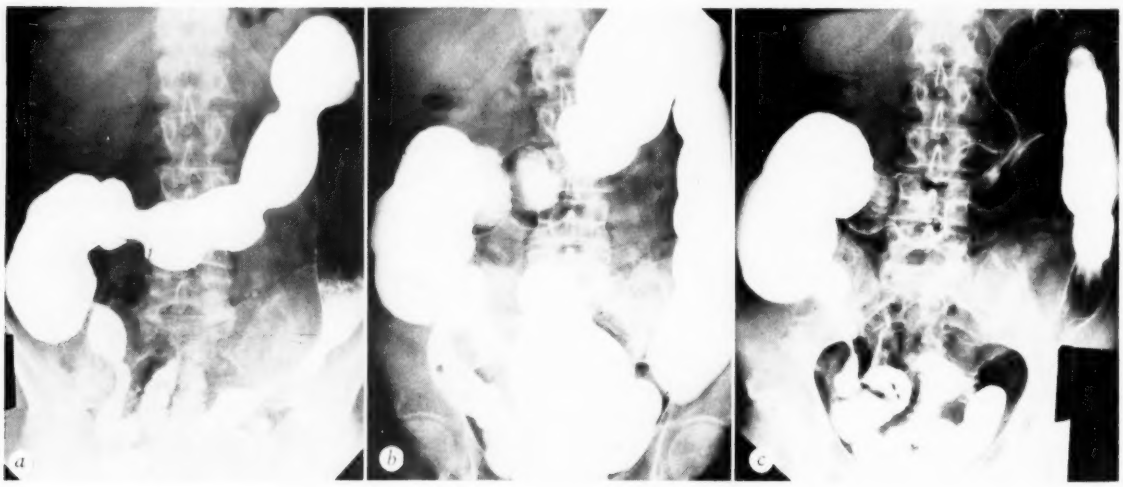

FIG. 6. (*a*) Postevacuation roentgenogram of colon showing lack of haustral markings and mucous membrane relief involving entire colon and terminal ileum. Roentgenograms of a filled colon (*b*) and after evacuation (*c*) at examination made eleven months later, during which time patient had been instructed not to use irritant cathartics. Appearance of colon has not changed since previous examination.

not included in this group gave a history of daily use, for a prolonged period, of several cascara wafers and 25 tablets of the proprietary cathartic the ingredients of which were listed previously. This patient clinically, proctoscopically and roentgenographically was diagnosed as having idiopathic chronic ulcerative colitis. One wonders if such extreme overdosage of irritant cathartics might have been the causative factor.

No definite evidence has been presented to indicate that the prolonged use of excessive doses of the irritant cathartics endangers the patient's life or reduces his longevity, except in acute poisoning with large amounts of the extremely irritating cathartic agents. The main reason for recognizing the changes described is to alert the clinician to the excessive use of irritant cathartics so that other, more physiologic means of bowel regulation may be proposed. Also, it is important for both clinicians and radiologists to recognize that prolonged and habitual use of the irritating group of cathartics can cause the development of a roentgenologic syndrome resembling mild or subsided colitis and enterocolitis, and thereby to avoid erroneous diagnoses, treatments and prognostications for the patient.

SUMMARY AND CONCLUSIONS

In 27 patients who gave clinical histories of prolonged, excessive cathartic usage, abnormal roentgenologic findings in the colon and terminal ileum consisted of loss of colonic haustrations, smooth mucous membrane as seen in relief, often apparent shortening of the right side of the colon, inconstant constriction of segments, and frequent marked distensibility of the colon. Such changes may be limited to the right side of the colon and terminal ileum, or may involve the entire colon. Their appearance may simulate the changes caused by chronic ulcerative colitis for which they have frequently been mistaken by the radiologist. These changes are caused by the irritant group of cathartics.

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THE INDUCTION OF A DISORDERED MOTOR FUNCTION PATTERN IN THE SMALL BOWEL BY THE ADMINISTRATION OF MINERAL OIL*

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THE demonstration of a disordered motor function pattern in patients in whom no reason could be found except that they were known constipates using mineral oil for corrective purposes prompted us to investigate the problem to determine whether the chronic use of mineral oil could be a valid etiologic factor.

Frazer *et al.*[1] experimentally found that hypertonic solutions (7 per cent sodium chloride, 10 per cent sodium bicarbonate or 40 per cent glucose) caused marked segmentation when introduced intraduodenally with the barium suspension. One per cent lactic acid or 0.3 per cent acetic acid given orally or intraduodenally also produced segmentation. Similar changes were seen by them using sodium oleate, olive oil (hydrolyzed to contain 28 per cent free fatty acid) and fecal fat recovered from a case of tropical and from a case of nontropical sprue.

Mineral fats are mixtures of hydrocarbons obtained from petroleum by fractional distillation and are either thick or thin, depending on the type of preparation. The mineral oils (the liquid petrolatums) consist mainly of naphthenes and polynaphthenes.

Histologic evidence shows that a small amount of oil is absorbed from the intestine in man as well as in rabbits, rats and guinea pigs, especially if the oil is emulsified with particles smaller than 0.5 micron. It is demonstrable in the mesenteric lymph nodes, liver and spleen.

For our studies we selected 100 individuals ranging from twenty-five to sixty years of age, who were free of signs or symptoms related to the gastrointestinal tract. A preliminary roentgenologic small bowel examination was performed on all, using the following routine: The patient was starved for twelve hours before the examination and then given 6 oz. of barium sulfate thoroughly mixed in tap water to make a 12 oz. suspension. Serial roentgenograms were obtained every half hour until the opaque material reached the cecum. Following this the patient was given 40 cc. of mineral oil by mouth daily at bed time for periods of between ten and twelve days. We then repeated the small bowel examination.

We realize that most patients taking mineral oil have done so for years and we accordingly would have hoped to continue administration beyond the twelve day period but found this difficult because these individuals were normal as regards their gastrointestinal tracts and were being discharged for their original conditions. No side effects were noted except an increase in bowel movements.

The 100 cases were divided into two equal groups. One group was given the regular barium sulfate mixture (Mallinckrodt) and the other was given micropaque (Picker) exclusively. The mixture contained 6 oz. of opaque material and tap water to make a 12 oz. suspension. We were unable to demonstrate any significant difference between the patients using the two types of barium in the preliminary studies. Some report that the regular chocolate barium produces some clumping, whereas micropaque does not.

RESULTS OF STUDY

The post-mineral oil medication series showed an incidence of 30 per cent exhibiting roentgen signs of a disordered motor

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function. The mechanism is not clear. It is possible that the oil separates from the opaque substance and thus produces a disordered motor function pattern or that the use of mineral oil over long periods of time (beyond that used in our studies) prevents the absorption of certain vitamins (D and K) and minerals (calcium and phosphorus), thereby causing a nutritional disturbance and subsequently a neuromuscular disorder of the intestinal wall.

CONCLUSION

A disordered motor function pattern in the small bowel can be induced in normal individuals by the administration of mineral oil and, accordingly, is certainly present in the chronic constipates who habitually use mineral oil.

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A STUDY OF FAT ABSORPTION IN THE PROXIMAL SMALL BOWEL*

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PREVIOUS investigations in this laboratory on fat absorption, using an I^{131} labeled triolein test meal following gastrectomy and/or gastroenterostomy, showed that many of these postoperative patients had abnormal fat absorption. Shingleton *et al.*,[3] showed that in a study of 50 cases of partial gastric resection the fecal fat recovery averaged 14.1 per cent as compared to 0.6 per cent for the normal person. This paper is concerned with furthering the understanding of the mechanism producing the abnormal test data in these subjects. The experiment is an attempt to duplicate this abnormality in a nonsurgical manner.

PROCEDURE

Thirteen healthy male Duke University students were used as subjects, after fasting for a minimum of twelve hours. A Miller-Abbott tube was introduced into the upper jejunum. The end of the tube was placed in the proximal jejunum past the site where a surgeon would perform a Bilroth II gastroenterostomy. This position was then confirmed by an abdominal roentgenogram. The test meal was administered after deflation of the balloon on the Miller-Abbott tube; it consisted of an emulsion containing one part peanut oil, one part water, and one twenty-fifth part Tween 80 as an emulsifier. The dose was 1 ml. emulsion per kilogram of body weight and to this was added 20 μc I^{131} labeled glycerol trioleate (obtained from Abbott Laboratories, Chicago, Illinois, under Atomic Energy Commission authorization). The meal was administered through the tube by a syringe. The syringe was washed twice and the wash administered following the meal. Two ml. venous blood samples were collected at one, two and three hours and placed in individual tubes. These were assayed in a sodium iodide, thallium activated, scintillation crystal well counter. The total blood volume was assumed to be 7.2 per cent of body weight. Using the measured radioactivity in the 2 ml. samples and a calculated total blood volume, the radioactive I^{131} blood level was determined and expressed as a percentage of the administered dose. The one, two, and three hour samples were added together and expressed as their sum. Urine free stool specimens were collected during the forty-eight hours following administration of the meal and placed in individual, plastic lined, one pint, waterproof cartons. The stools were assayed with a scintillation crystal detector and a Nuclear Instrument and Chemical Company Model 161-A scaler. The detector was positioned 30 cm. from the top surface of the sample container. The samples were counted for a sufficient time to give statistical accuracy. These were then compared to a 100 gm. homogeneously distributed I^{131} standard, equivalent to that ingested by the patient. Fecal recovery was expressed as a per cent of the total administered dose.

RESULTS

Table I shows the results obtained from the intubated experimental subjects listed as individual hourly blood concentration, sum of the one, two and three hour blood samples, and total fecal recovery. All are expressed as a per cent of the total administered dose. These are averaged and their standard deviation computed. Table II compares the Bilroth II group reported by Shingleton *et al.*[3] and the intubated subjects with the normal values or the orally administered test meal.[1]

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The technical assistance of Miss Elizabeth Kent and Mrs. Kathryn Sharpe is gratefully acknowledged.

TABLE I

Subject Number	Blood Levels (per cent)			Sum (1, 2, 3 hr.)	Fecal Recovery (per cent)
	(1 hr.)	(2 hr.)	(3 hr.)		
1	0.6	2.8	2.5	5.9	56.0
2	2.3	3.0	3.9	9.2	28.0
3	0.7	1.1	1.6	3.4	57.0
4	2.5	4.1	5.3	11.9	49.0
5	0.7	1.0	0.9	2.6	64.0
6	1.8	1.7	1.9	5.4	72.0
7	0.2	0.2	0.4	0.8	5.4
8	0.0	0.6	0.0	0.6	86.0
9	0.1	0.5	0.1	0.7	88.0
10	2.0	2.6	2.6	7.2	67.0
11	0.4	0.5	0.4	1.1	46.8
12	1.1	0.9	1.2	3.2	42.5
13	0.8	1.0	1.3	3.1	82.7
Average	1.0	1.5	1.7	4.2	57.2
Standard Deviation	0.856	1.21	1.55	2.03	23.6

DISCUSSION

This experiment is an attempt to determine why the postgastrectomy patient does not absorb the radioactive fat meal to the same degree as the normal person. Bypassing the duodenum and first portion of the jejunum via a Miller-Abbott tube seemed to be the best way to demonstrate the part played by this area of the gastrointestinal tract in the digestion or absorption of fat.

In a previously reported study[1] it was found that the highest blood values of radioactivity occurred at four, five and six hours, concomitant with the passage of the orally administered test meal through the small intestine. In this study, blood samples were drawn at one, two and three hours because the test meal was introduced directly into the small intestine. It is thought that this makes them comparable to the four, five, and six hour samples in the orally administered test.

TABLE II

	Normal Controls	Bilroth II Subjects	Intubated Subjects
Average Blood Value	40.3	24.8	4.2
Average Fecal Value	0.6	14.1	57.2
Standard Deviation Blood Value	9.6	8.6	2.0
Standard Deviation Fecal Value	0.4	16.8	23.6
Number of Subjects	25	50	13

Table II shows the average blood value in the normal subject to be the highest and that of the Bilroth II group and intubated subject to be progressively lower. The blood levels decrease as more proximal small bowel is bypassed. It is observed also that the fecal fat recovery in the normal control is the lowest, with a successive rise in the Bilroth II and intubated subject. Fecal recovery rises as more proximal small bowel is bypassed by the test meal. These data confirm that the degree of intestinal absorption of this test meal is dependent upon a function of the proximal small intestine. Absorption of the meal decreases as more of the upper small bowel is bypassed by the meal.

We interpret these data to mean that either a large portion of fat absorption oc-

curs in the duodenum and jejunum,[2] or some necessary event prior to absorption occurs as a result of the meal passing through the duodenum and jejunum.

SUMMARY

Thirteen healthy experimental subjects were intubated to the level of the proximal jejunum past the site of the gastroenterostomy of a Bilroth II gastric resection. An I^{131} triolein test meal was administered through the tube. Blood samples and feces were assayed for radioactivity and compared to a group of 25 normal controls and a group of 50 postoperative Bilroth II gastrectomy patients. Thus 2 groups of subjects are presented with progressively more proximal small bowel bypassed by the test meal. It was demonstrated that as more proximal bowel was bypassed by the test meal less fat was absorbed and more fat was excreted in the feces.

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OBSERVATIONS ON THE ABSORPTION OF UROKON FROM THE PATHOLOGIC GASTRO-INTESTINAL TRACT*

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THE aqueous organic iodide contrast media have found wide acceptance for use in pyelography and angiography and, more recently, in gastrointestinal tract roentgenology as well. Clinical experience with these compounds in roentgen examination of the gastrointestinal tract has been attended by occasional unexplained evidence of gross absorption[1,2] and has stimulated us to an independent study of the absorption of one of these compounds (urokon) from the alimentary tract. It was observed that approximately 20 per cent of an orally administered tracer dose of radioactive urokon is excreted in the twenty-four hour urine specimen of normal individuals, but a tenfold increase in the amount of urokon ingested resulted in an almost proportionate decrease in the percentage excreted.[3] It appeared that the normal bowel functions as a barrier to pronounced absorption, suggesting that gross absorption is attended by disruption of this barrier by bowel pathology. Accordingly, a study was undertaken to determine the absorption of radioactive urokon in patients with gastric and colonic malignancy, bowel fistulae, and in patients treated with pelvic irradiation for carcinoma of the cervix.

A total of 24 patients in the above categories ingested 50 cc. of radioactive urokon; they were selected by excluding only those with known iodide sensitivity, history of recent iodide intake or with abnormality of urinary tract and thyroid gland. Medications and enemas were withheld for twenty-four hours before and after the start of the study and subjects fasted for nine hours before and three hours after the oral ingestion of urokon. The radioactivity of the twenty-four hour urine sample was determined in terms of the percentage of urokon administered, and the results compared with that found in the normal (Table 1).

TABLE I

Pathology	No. of Cases	24 Hour Urinary Excretion
		(per cent)
None	4	20.9
Gastric Carcinoma	6	8.4
Carcinoma of Colon and Rectum	4	21.2
Bowel Fistulae	3	36.2
Radiation Therapy to Abdomen	8	34.1
Obstructive Jaundice	3	38.6

All 6 patients with gastric carcinoma manifested a marked decrease in absorbtion of urokon from the intestine. Four of these patients had active bleeding from ulcerating malignancies, thus indicating that the presence of a bleeding point in the gastrointestinal tract is not necessarily associated with increased absorption. The mechanism of decreased absorption in the presence of gastric carcinoma is yet to be determined. None of these patients manifested pyloric obstruction on barium studies, excluding gastric retention as a cause of decreased absorption. Achlorhydria was present in 4 of these patients and may bave played a part in the decreased absorption which they manifested. Substance is given to this possibility by our observation that in normal patients a 40 per cent decrease in absorption is effected by alkalinization of the orally administered radioactive urokon with 4 gm. of sodium bicarbonate. An additional factor may be that gastric absorption is reduced following mucosal de-

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struction by gastric malignancy. In dogs, radioactive urokon was introduced into the isolated stomach with 5 per cent excretion occurring in the three hour urine specimen, thus establishing that absorption from the normal stomach does occur.

In the patients with uncomplicated carcinoma of the colon and in 2 patients with gross rectal hemorrhage from carcinoma of the rectum, absorption was normal.

Two patients who presented with fistulous tracts between sigmoid and ileum, secondary to sigmoid malignancy, evidenced a marked increase in absorption from the gastrointestinal tract. A similar finding was noted in a patient with a large walled-off sinus tract, secondary to a perforated diverticulum of the left colon. The rapid absorption of aqueous organic iodide contrast media from sinus and fistulous tracts is well known, and the use of orally administered radioactive urokon for their detection would appear to have merit.

Eight patients with carcinoma of the cervix, treated with intravaginal and external radiation therapy were studied. All patients evidenced an increased absorption of radioactive urokon from the intestine, suggesting a disruption of the mucosal barrier to absorption as a result of bowel irradiation.

Three cases with obstructive jaundice were studied and all evidenced the unexpected finding of increased absorption. The diagnoses in these cases were: (1) carcinoma of the common duct without hepatic metastases; (2) carcinoma of the colon with hepatic metastases; and (3) cholelithiasis and cholangitis.

To substantiate these findings, radioactive urokon was introduced into the small bowel of 4 dogs, seventy-two hours after ligation of the common duct. In 3 of these dogs absorption was increased from 50–100 per cent above that noted in the control animals; in the fourth, no increase was noted. To explain this increase, it was postulated that normally urokon absorbed from the small intestine is re-excreted in large amounts into the bile. In the presence of obstructive jaundice, re-excretion would no longer occur. To test this hypothesis, the common duct of 2 dogs was catheterized and the radioactivity of the bile compared to that of the urine following the oral administration of radioactive urokon. At three hours, the urine contained 12.7 per cent of the orally administered urokon, and the bile less than 1 per cent, thus invalidating this hypothesis. It is considered possible that bile acting as an anionic surfactant complexes with the urokon molecule to retard its absorption. In the absence of bile, absorption is, therefore, enenhanced.

SUMMARY

The absorption of radioactive urokon from the gastrointestinal tract is altered from the normal in several varieties of bowel pathology. Absorption was found to be markedly diminished in gastric carcinoma and considerably increased in patients with obstructive jaundice, bowel fistulae and in patients subjected to bowel irradiation. Absorption was not altered in patients with malignancy of colon or rectum. The presence of a bleeding point in the gastrointestinal tract did not result in increased absorption.

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ORALLY ADMINISTERED WATER SOLUBLE IODINATED CONTRAST MEDIA*

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WATER soluble iodinated contrast media constitute an indispensable part of our standard armamentarium in diagnostic radiology. Suitably prepared and dispensed, they have enabled us, during the past third of a century, to explore the urinary tract, the cardiovascular system, the biliary tract, as well as special areas of the body where fistulous tracts, sinuses and various cavities may be found.

In the gastrointestinal tract, the most readily accessible system of the body, traditionally, nonwater soluble contrast media have been in use. Bismuth subnitrate, soon replaced by bismuth subcarbonate, was used as early as 1897.

Barium sulfate, originally suggested by Cannon in 1904, did not come into use until 1910.[1] Since then, chemically pure barium sulfate, with slight modifications in its physical state (colloidal barium, micropaque) has remained virtually unchallenged as the contrast medium for gastrointestinal examination. Its use has become so firmly entrenched in our daily routine that all our techniques and procedures have developed and actually revolve around this contrast medium.

Barium sulfate has the advantage of being readily available, inexpensive, nontoxic, and nonabsorbable. It produces a shadow of consistent density with sharp delineation of the surrounding parts. The taste is not objectionable, though not pleasant. On the other hand, it is not water soluble; its particles remain discrete and tend to precipitate; it is unsuitable for outlining minute fistulous tracts; and has a great tendency to inspissate after water has been absorbed, especially from the colon. Thus, barium impactions occur which at times result in obstruction (Fig. 1). Unfavorable consequences may follow when it is accidentally introduced into other body cavities, *e.g.*, the peritoneum or the tracheobronchial tree.

FIG. 1. No. 6852, a fifty-two year old male. Routine gastrointestinal series performed elsewhere. Barium impaction in descending colon with complete proximal intestinal obstruction. Surgical removal of impaction relieved the obstruction. No intrinsic lesion of the colon was found.

Thus, the need for an improved contrast medium for the examination of the gastrointestinal tract is quite obvious. Such a contrast medium should meet the following criteria. It should be:

(1) palatable;
(2) nonabsorbable from the lumen of the stomach or intestines;
(3) nontoxic, with absence of local or sys-

* Presented at the Ninth International Congress of Radiology, Munich, Germany, July 23–30, 1959.

temic reaction. If accidentally introduced into other body cavities it should cause no toxicity or irritation;

(4) sufficiently dense to insure consistent roentgen visualization with accurate detail;

(5) water soluble, retaining the same consistency at all times, with no precipitation;

(6) low in viscosity. This permits it to flow freely, with uniform distribution, to conform to the outline of the viscus or cavity into which it is introduced;

(7) fluid enough to pass through narrow areas and fistulous tracts;

(8) inert with no disturbance of the normal anatomy and physiology of the parts with which it comes in contact;

(9) readily and completely eliminated from the gastrointestinal tract;

(10) inexpensive.

Recently, considerable interest has developed in the use of water soluble iodinated contrast media in the examination of the gastrointestinal tract. Appreciable progress has been attained in this direction. This has been made possible by the adaptation of contrast media used in the examination of the urinary tract.[2,3,4]

MATERIAL

This report is based on our experience with 200 patients examined, 100 each with hypaque (hypaque sodium, with 0.1 per cent polysorbate 80, Winthrop) and gastrografin (oral renografin 76 per cent, Squibb).

These media were used alternately on all patients referred to our department for routine examination of the gastrointestinal tract. This afforded an opportunity to study the normal as well as the abnormal, to evaluate and standardize the technique for this type of examination, to observe the anatomy and physiology of the gastrointestinal tract as manifested by the use of these contrast media, and to assess the advantages and disadvantages, the scope of usefulness as well as the limitations. At this time, however, chiefly due to cost and taste, these media are used in cases selected from our daily work load.

Hypaque (diatrizoate sodium) is available in powder form, dispensed in a metal container and provided with a measuring spoon. The optimal preparation consists of one measuring spoon of powder and two of water. Four to six ounces are adequate for a single examination. Greater or lesser concentrations can be easily prepared by changing the ratio of the powder and the water. This preparation has the advantage of being compact and having nonbreakable containers, which are convenient for storage.

Gastrografin is a solution of diatrizoate methylglucamine 76 per cent. It is dispensed in amber colored bottles containing 120 cc. of the solution. It has the advantage of being prepared in advance and is ready for use without further preparation. It is lemon flavored in an attempt to mask the taste. However, the bottles are breakable and occupy a relatively large space in storage.

Hypaque sodium or gastrografin may be dissolved in ginger ale. The taste of the ginger ale masks the bitterness of the basic ingredients and gives the added advantage of a double contrast effect resulting from liberation of the carbon dioxide gas.

Both preparations are practically identical in chemical behavior and in their pharmacology. Both remain indefinitely in solution. The addition of a wetting agent or Tween insures "cohesiveness" and better delineation of the mucosa. They neither precipitate nor settle on standing as does barium sulfate and micropaque. Animal studies reveal absence of any toxicity. This is confirmed by the experience of other workers as well as our own. No absorption was noted in any of our patients and we have not seen any roentgenologic evidence of urinary excretion. However, traces of iodine excretion have been recovered in the urine. In 2 cases reported by other investigators, sufficient urinary excretion occurred, as to result in visualization of the urinary tract.[3]

CLINICAL ROENTGENOLOGIC OBSERVATIONS

Administered by mouth both hypaque sodium and gastrografin in solution pass rapidly through the esophagus, which is well visualized and satisfactorily outlined (Fig. 2). However, they are so thin in consistency that they fail to adhere to the esophageal mucosa. Thus, a complete investigation of the mucosal pattern cannot always be obtained. Techniques for overcoming this are being evaluated in our department at present.

The gastric and duodenal mucosa is excellently delineated (Fig. 3, *A* and *B*). While the density of the shadow is a little less than that obtained by barium, the normal and abnormal mucosa, ulcers of the stomach or duodenum (Fig. 4, *A–C*) as well as tumors are excellently demonstrated. Perfusion through and around clots in a bleeding esophagus, stomach or duodenum, is readily attained.

In the small intestines the shadow becomes less dense. The further away from

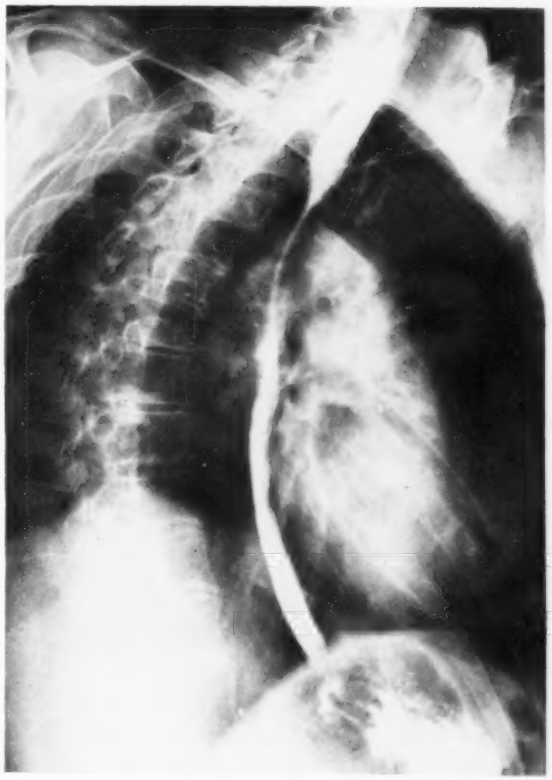

Fig. 2. Routine visualization of normal esophagus.

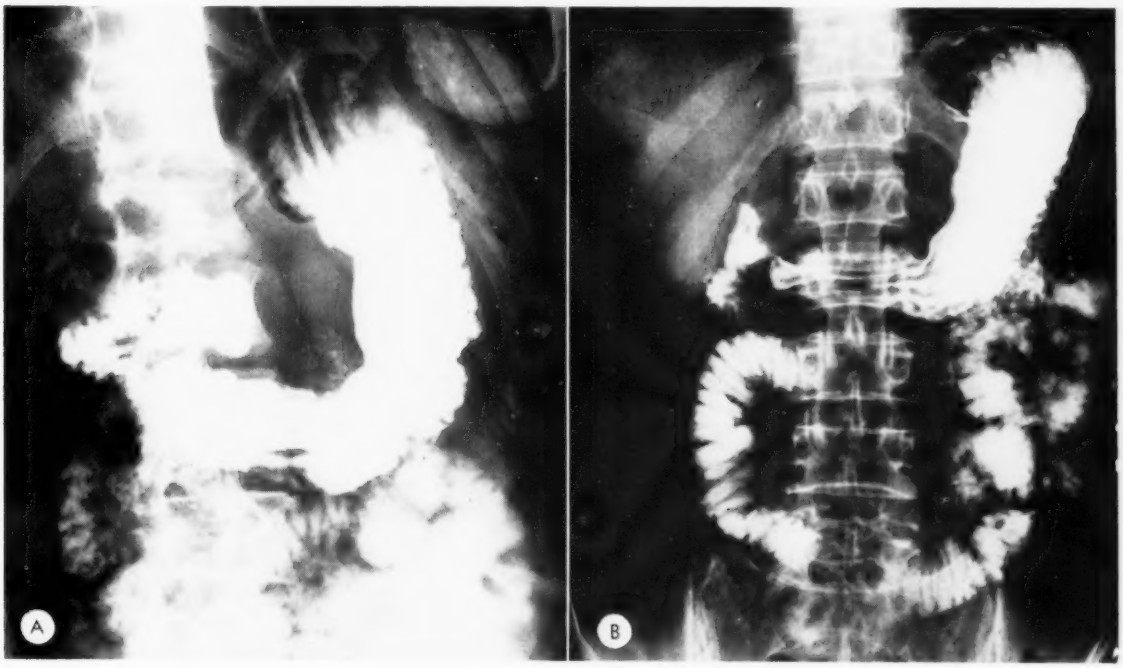

Fig. 3. (*A* and *B*) Normal stomach and duodenum.

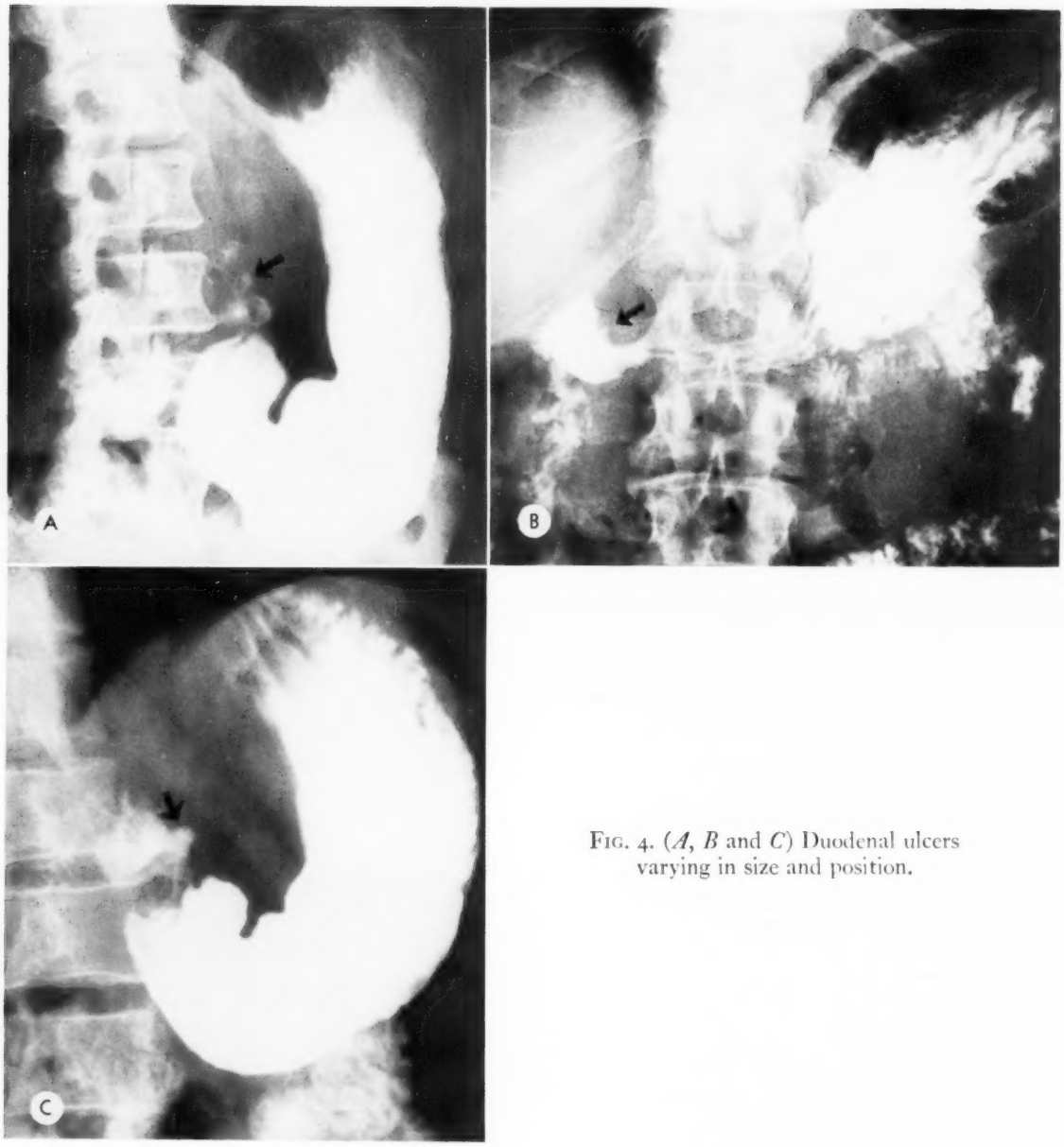

Fig. 4. (*A*, *B* and *C*) Duodenal ulcers varying in size and position.

the pylorus, the more dilute the solution becomes and the less clear the definition of the small intestinal folds. Thus the diagnostic quality is rendered inadequate as compared with barium sulfate (Fig. 5, *A* and *B*). Special areas of the small intestines may be studied by administering small amounts of the contrast medium through a previously introduced Miller-Abbot tube.

In the colon, where absorption of water occurs, the density of the contrast medium improves, resulting in excellent delineation of the entire colon. The contour is clear, haustral markings are well defined, almost equally well following administration by rectal enema as by mouth. Visualization of the colon is almost as good as with a barium enema.

The emptying time of the stomach and the transit through the intestines are accelerated. Gastric evacuation is complete within one to two hours. Within four hours

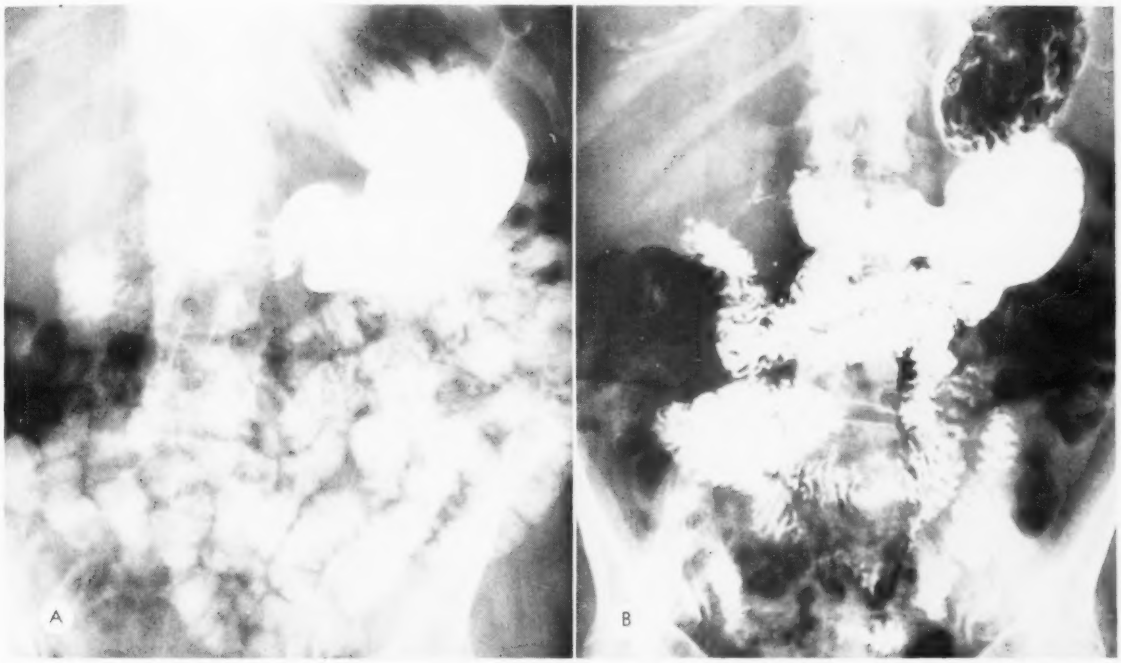

Fig. 5. No. 6587. A 48 hour interval between examinations. (*A*) Roentgenogram made immediately after oral administration of a water soluble iodinated contrast medium shows rapid and copious flow into small intestines. (*B*) Roentgenogram after oral administration of barium sulphate demonstrates sharp definition of small intestinal pattern.

all the contrast medium is usually out of the small intestines, delineating the entire colon (Fig. 7, *A–D*).

Despite the rapid transit, no diarrhea has been observed in our cases. There was no evidence of sensitivity to the contrast medium whatever. Precipitation or crystallization of the salt has been seen in the stomach at two to three hours where there has been apparent prolonged retention (Fig. 6, *A* and *B*). Sometimes the colon shows the same type of precipitation (Fig. 8). The significance of this phenomenon is not yet known to us and apparently there are no accompanying untoward effects.

Table 1 gives the significant findings observed in our series of 200 cases. Our experience with routine enema studies of the

Table I

ORALLY ADMINISTERED WATER SOLUBLE IODINATED CONTRAST MEDIA; FINDINGS IN 200 NONSELECTED CASES

Esophagus—hiatal hernia	9
Fibrosis and incomplete obstruction of distal esophagus	1
Negative examination of the gastrointestinal tract	95
Pylorospasm without demonstrable cause	7
Gastric carcinoma	3
Benign polyps of the antrum	2
Gastric ulcer, benign	3
Gastritis	5
Pyloric obstruction	7
Pyloric ulcer	1
Duodenitis	9
Duodenal ulcer—anterior wall	19
Duodenal ulcer—posterior wall	9
Duodenal bulb deformity without demonstrable crater	13
Prolapse of the gastric mucosa through the pylorus	9
Small intestinal obstruction	
Incomplete	3
Complete	5
	200

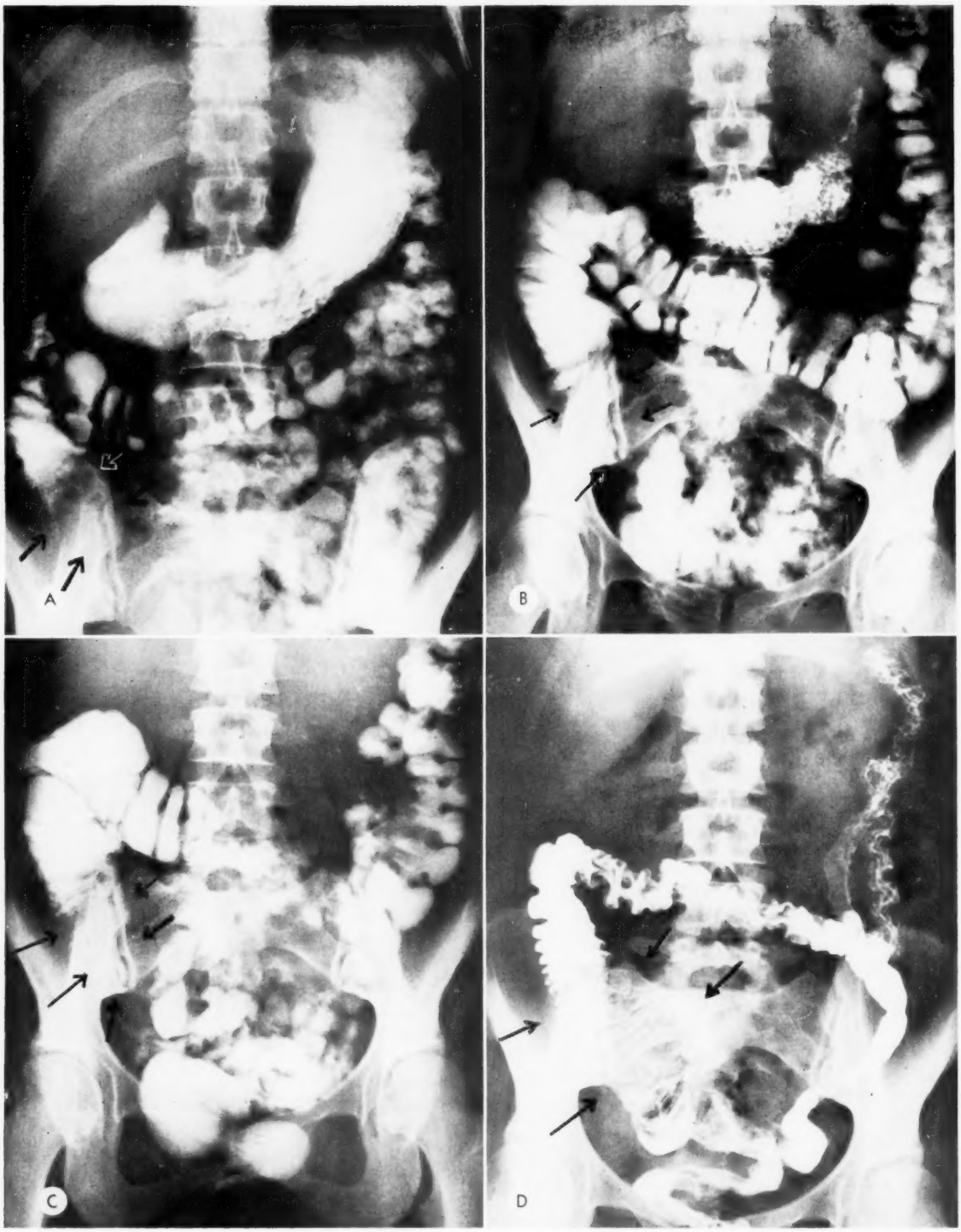

Fig. 6. No. 8239. Iodine precipitation at (*A*) 1 and (*B*) 4 hours with prolonged gastric retention. Note defect at base of cecum and region of terminal ileum. (*C*) Clinically there was a palpable mass, confirmed by enema studies, revealing pronounced intussusception on postevacuation roentgenogram (*D*).

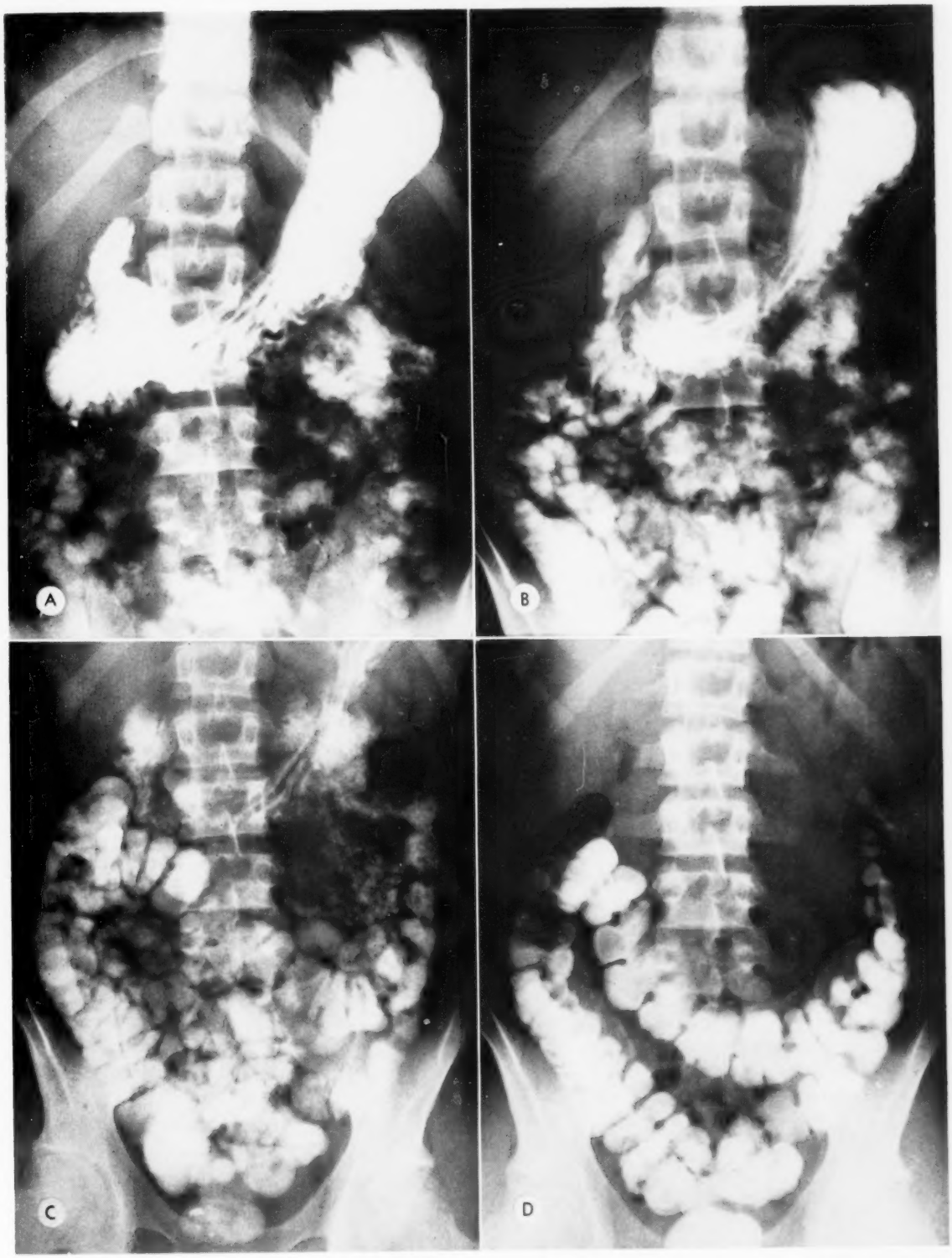

Fig. 7. (*A–D*) Roentgenograms made at 5 minutes, 30 minutes, 1½ hours and 4 hours. Almost complete gastric evacuation is noted at 1½ hours. At 4 hours the colon appears well outlined with no contrast medium in the stomach or small intestines.

Fig. 8. No. 5652. Iodine precipitation in colon, 24 hour roentgenogram.

colon is still limited (Table II). We can say at this time that delineation of the colon is as satisfactory as it is with barium. Emptying of the colon, however, is often not quite complete. What seems to be complete obstruction to barium proves to be partial with these water soluble media, thus affording a good opportunity to localize and delineate the offending cause or lesion.

The rapid transit of the contrast medium through the gastrointestinal tract presents considerable saving of time to the patient as well as to the department and a small, but significant saving in the cost of films used.

None of our patients experienced any nausea, vomiting, diarrhea or showed any evidence of toxic reactions whatsoever.

Inasmuch as the contrast medium reaches and outlines the entire colon within four to six hours, further follow-up through a twenty-four-hour period is not necessary. The hospital stay of in-patients is thus shortened and out-patients are spared the necessity of returning for additional visit on the following day.

The taste, despite various forms of masking, remains bitter and is only slightly improved. The patient, if informed beforehand of the taste and of what to expect, will cooperate more adequately than he would otherwise. We have had no difficulty in this connection.

Table II

ENEMA STUDIES WITH WATER SOLUBLE IODINATED CONTRAST MEDIA; COLON FINDINGS IN 13 SELECTED CASES

Normal	5
Colitis	1
Carcinoma	2
Volvulus	1
Intussusception	1
Obstruction—cause undetermined	3
	13

CONCLUSION

Water soluble iodinated contrast media in the form of hypaque sodium and gastrografin present a great advance in the contrast studies of the gastrointestinal tract and mark a milestone of progress in the direction of attainment of a perfect contrast medium for this purpose.

It meets most of the criteria stated above but fails as to taste and cost.

We are coming closer to the day when we shall be freed of the fear of barium impactions, when these contrast media, or an improved successor, will be readily available and widely used at less cost to the patient and with greater delight to his taste buds. Only then will either or both products replace barium sulfate in the routine examination of the gastrointestinal tract.

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THE USE OF CINEFLUOROGRAPHY IN THE ROUTINE DIAGNOSIS OF DISEASE OF THE UPPER GASTROINTESTINAL TRACT*

EXPERIENCE IN 1,000 CASES

By JOSEPH JORGENS, M.D., PH.D., ILMAR O. KIESEL, M.D., *and* HARLAN W. HAWKINSON, M.D.

MINNEAPOLIS, MINNESOTA

THE purpose of this paper is to present our evaluation of cinefluorography in the routine diagnosis of disease of the upper gastrointestinal tract. Because the idea of motion picture roentgenography has been conceived as the ultimate in roentgen diagnosis of the gastrointestinal tract, cinefluorography has great appeal to radiologists. It is, however, necessary to admit that the cinefluorograph is not the absolute answer in the diagnosis of cancer nor even entirely dependable in detecting an ulcer. This conclusion has been forced on us by our experience of the last two years with over 1,000 examinations of the upper gastrointestinal tract at the Minneapolis Veterans Administration Hospital. We have been using the cinefluorograph (Westinghouse Cine Fluorex) in much the same manner as we use our standard fluoroscope. A report of several uses and certain disadvantages of cinefluorography in clinical practice might help the potential buyer and user of this equipment.

EQUIPMENT

Our cinefluorograph consists of a standard Westinghouse fluoroscopic table equipped with a Westinghouse image amplifier and a motion picture camera which uses 16 mm. film and exposes either 15 or 30 frames per second. The image amplifier brightens the fluoroscopic image so that it can be photographed.

FILM PROCESSING

We use Kodak Linagraph Shellburst film, packaged in 100 foot rolls. After exposure, the role of film is wrapped about a stainless steel square which will fit the standard 20 gallon developing tank (Fig. 1). Each corner of the square consists of $1\frac{3}{16}$ inch of stainless steel tubing.* This frame for developing will hold 100 feet of film. Special solutions are not required. Developing our own film has proved more

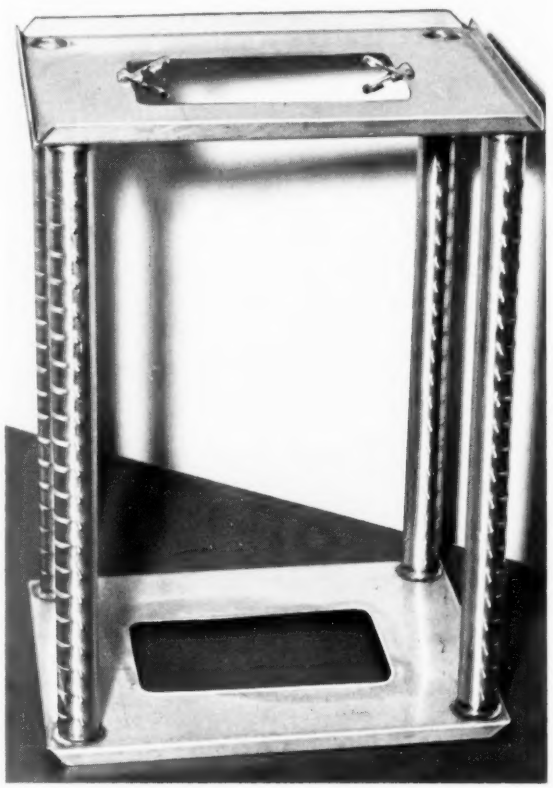

FIG. 1. One hundred foot roll of film can be wrapped about this stainless steel frame which can be placed in a standard 20 gallon developing tank.

* Cronstroms Manufacturing Inc., 4225 Hiawatha Avenue, Minneapolis, Minnesota.

* Presented at the Sixtieth Annual Meeting of the American Roentgen Ray Society, Cincinnati, Ohio, September 22–25, 1959.

convenient and we obtain a quality as good, if not better, than that obtained at local commercial processors.

CLINICAL MATERIAL

All of our patients were adults, mostly males, the great majority being between thirty-five and seventy years of age. This age range is important since there is the question of radiation hazard associated with cinefluorography.

TECHNIQUE OF THE CINEFLUOROGRAPHIC EXAMINATION

The usual fluoroscopic examination can be carried out without much change in habits. However, it is necessary to sit on the right side of the patient since the image amplifier tube is in front of the fluoroscopic screen. Unless you have a long arm, it is more convenient to palpate the patient with the standard fluoroscopic cone. Spot roentgenograms are taken in the usual manner. The cinefluorographic recording is also simple to make as only one lever is moved. Adjustments of the exposure factors for the motion picture recording are effected automatically.

We have substituted the cinefluorogram for most spot roentgenograms. However, we still take the conventional post-fluoroscopic roentgenograms of the stomach and duodenum as these portray better the relative size and position of organs.

RADIATION DOSAGE DURING CINEFLUOROGRAPHY

The over-all dosage of a cinefluorographic examination is dependent upon the output of the image amplifier and the cinefluorograph. The image amplifier delivers a smaller dose of radiation per minute than the conventional fluoroscope. The cinefluorographic radiation dose per minute is relatively high, especially when the abdomen is being recorded.

Our cinefluorographic unit operates at between 8 and 30 ma. If the entire upper gastrointestinal tract were cinefluorographed, the total air dosage would be less than 120 r with no one area receiving more than 50 r in air.

Rather than surveying the entire upper gastrointestinal tract cinefluorographically, we substitute the cinefluorogram for spot roentgenograms. This reduces considerably the amount of cinefluorographic time and radiation exposure.

USES OF CINEFLUOROGRAPHY

Esophagus. Cinefluorography can be recommended for the study of the process of deglutition.[4] The rapidity of the muscular movements during swallowing makes it impossible to record the serial changes on static roentgenograms. Furthermore, most radiologists have considerable difficulty trying to time their spot film exposures during the swallowing process. The changing phases of the swallowing process are readily captured on a single cinefluorogram.

In our department all swallowing problems are studied by means of cinefluorography. We usually take films at 15 frames per second. However, if we wish to study the swallowing process in slower motion, the cinefluorograph can be set instead to take 30 frames per second.

We have seen many interesting alterations in the swallowing process, including asymmetric muscle movement, regurgitation of barium into the nasopharynx following poliomyelitis,[1] fixation of the walls of the pyriform sinus and larynx due to invasion by carcinoma, and aspiration of a contrast agent into the upper larynx due to muscular dysfunction.

In the mid-esophagus there is no particular advantage in using the cinefluorograph. In fact, the restricted field size of the image amplifier may be a disadvantage.

In the lower esophagus the cinefluorographic method is again advantageous to use as fairly rapid changes in the shape and size of the esophagus occur. Although our understanding of the conditions associated with regurgitation of barium from the gastric fundus into the lower esopha-

gus has increased, no new entities have been discovered. The cinefluorograph may be of greater value than standard fluoroscopy in detecting early malignant lesions of the esophagus.

Objectively, the degree of accuracy of detection of varices by the cinefluorographic examination is difficult to establish since the endoscopic and histologic detection of varices is limited in accuracy. Nevertheless, because varices seem to be detected easier with the cinefluorograph, all patients who are suspected of having varices of the esophagus are now examined here cinefluorographically. The partially relaxed state of the esophagus appears to be the best stage for detecting varices. In the great majority of cases the maneuvers of Valsalva and Müller have been impossible to carry out because of the mental and physical condition of our patients.

No real limitation of recording the esophagus cinefluorographically has been noted, except for the small field size of the Cine Fluorex. However, conventional roentgenograms usually provide necessary information about positional relationships of organs.

Stomach. A cinefluorographic study of the body and fundus of the stomach does not add anything further to the diagnostic findings obtained on conventional spot roentgenograms. However, in the antrum of the stomach the structure and peristaltic function are better recorded by cinefluorography than by spot roentgenography. Not only can slight abnormalities in the structure of the stomach wall be more easily detected under the stress of a peristaltic rush, but we can be more confident that these findings are actually present.

Interesting minor changes, previously unrecognized, such as the abnormal movement of the mucosa, apparently associated with the fixation of the mucosa to the underlying muscularis propria, are now being followed to determine their possible significance.

In the antrum a slight alteration in the character of the peristaltic movement itself may suggest a diseased state. For example, a distorted peristaltic movement may be caused by an adhesion of the stomach wall to the adjacent inflamed pancreas. A retrograde peristaltic movement may occasionally be seen in the presence of an ulceration in the distal antrum, or a localized, slightly rigid wall may suggest the presence of an inflammation or tumor of the stomach wall.

Duodenal Bulb.[2] The cinefluorographic study of the duodenal bulb is extremely useful. Its size and shape can be recorded during its entire contraction and expansion sequence. Many times a shallow ulcer will only appear during a certain phase of the contraction, and consequently can be missed by spot film techniques. In addition, in obese individuals or in those with ascites, the bulb, often located high under the ribs, is difficult to palpate. The diagnostic value of conventional fluoroscopy is, at best, poor in such individuals. By means of cinefluorography a fairly good examination of the bulb can be obtained. The image amplifier gives a bright detailed image of the bulb and the motion picture camera can record the entire contraction-expansion sequence.

There are two frequent normal types of bulb contraction, one type occurs in the mid-segment, initiated apparently by overdistention of the bulb, and the second type consists of a contraction of the proximal portion of the bulb. This usually is observed near the end of the systolic contraction of the antrum. Variations of these types of contractions of the bulb are noted often associated with strictures, ulcers of the base of the bulb, and deformities resulting from previous ulcers. There is no better way of evaluating these abnormal peristaltic patterns than through the method of cinefluorography.

DISCUSSION

Our cinefluorograph, the Westinghouse Cine Fluorex, has proved practical in our

hospital for routine use. We feel that the information gained through cinefluorography is worth the extra effort and money involved.

To make the method practical and stay within a limited budget, we found that we could process the Kodak Linagraph Shellburst film in the department using standard solutions and tanks. The only extra pieces of equipment we have had to buy were several stainless steel square frames onto which the 100 feet of film is wrapped and processed. We allow the film to dry in open air rather than in a hot air dryer. Hot air tends to make the film brittle. However, if necessary, the film can be dried within an hour in a commercial hot air dryer. By this conventional processing, a negative is obtained which shows the barium as white and the air as black. Our 16 mm. film requires no reduction in size or extra prints made, unlike the system now used in processing 35 mm. film.[3]

A projector which projects an image a distance of 4 to 8 feet and which has an instantaneous reversing feature is sufficient and adequate for diagnostic purposes. A flat white illustrator type cardboard can be used as a screen and is, in fact, superior to an expensive beaded glass screen.

We found, from a practical standpoint, that the cinefluorographic film replaces the need for a number of static spot roentgenograms. Whereas we formerly took many spot roentgenograms to record the diseased area to advantage, now two film strips may be all that is necessary.

The cinefluorographic study has a real advantage over the conventional study in one recurring situation. Frequently, patients do not hold their breath because of respiratory conditions, mental disturbance, or difficulty in understanding instructions. In such situations it is almost impossible to obtain satisfactory spot roentgenograms or even conventional post-fluoroscopic roentgenograms. The whole examination is often unsatisfactory due to this lack of cooperation. In contrast, the cinefluorographic study is unaffected by these factors and, therefore, of definite advantage in these instances.

At present there are several disadvantages to cinefluorography which should be considered by those contemplating its use.

The cinefluorograph is heavier and more cumbersome to manipulate than the conventional fluoroscope. Actually, the image amplifier is the heaviest part of the cinefluorograph. The camera is relatively small and light. A power assist has been added to the cinefluorograph which aids somewhat in vertical movement, but the image amplifier is still quite heavy. This means that the cinefluoroscopist must be willing and able to manipulate the instrument through a morning's work. It would be wise for any potential buyer to try out the cinefluorograph of his choice in the field before he purchases it. For most women radiologists, the cinefluorograph is a little too heavy. The cinefluorograph we use has a small field size, 4.7 inches in diameter. For a good fluoroscopist this is only occasionally a disadvantage as most experienced fluoroscopists survey only a small field. The relative sizes and positions of organs can be recorded on standard roentgenograms following the cinefluorographic examination.

The cinefluorograph is expensive. The Westinghouse image amplifier with camera costs about $15,000 and this is in addition to the price of a good standard fluoroscopic unit to which this amplifier and camera are attached. The cinefluorographic film is more expensive than the spot film which it replaces. The cost of 33 feet of 16 mm. film is about $1.35. The cost of four spot films, 8×10 inch in size, is about 76 cents. Besides this, the repair and maintenance of the equipment is at least double that of the conventional fluoroscope.

CONCLUSION

The cinefluorograph consists of three essential parts: a standard fluoroscopic unit,

an image amplifier and a motion picture camera. In the study of the gastrointestinal examination the chief advantages of the cinefluorograph are: (1) the image amplifier provides a bright and detailed fluoroscopic image, (2) the camera records the characteristics of the peristaltic wave which may be of prime importance in the early detection of disease of the gastrointestinal tract, and (3) the motion picture film provides a permanent record of the fluoroscopic examination for study and reference.

The main disadvantages of the cinefluorograph are: (1) it is somewhat heavy and awkward to handle, (2) the field size is small, and (3) it is expensive to buy and maintain.

In spite of these few disadvantages the Westinghouse Cine Fluorex has proved practical at this thousand bed Veterans Hospital.

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EDITORIALS

RADIOLOGIC ASPECTS OF THE MALABSORPTION SYNDROME

THE increasing frequency with which the malabsorption syndrome is being recognized as a primary entity or as a secondary condition to some systemic disease process accelerates the search for simple, accurate and inexpensive means of evaluation. Furthermore, modern advances in medicine make accurate diagnosis of importance from both a therapeutic and prognostic standpoint. A number of tests such as the vitamin A tolerance test, oral glucose tolerance test, vitamin B_{12} absorption test, iron tolerance test, I^{131} tagged fat absorption tests, determination of serum carotine level and d-zylose absorption studies have now acquired definite clinical significance in the evaluation of this syndrome. The roentgenologic examination of the gastrointestinal tract, particularly the small bowel, may also prove to be of considerable aid. More recently a serum turbidity test[1] following fat meal ingestion has been described.

Diseases which manifest malabsorption primarily are celiac disease of childhood, tropical sprue, and nontropical sprue. Regional enteritis, common bile duct obstruction, chronic pancreatitis, extensive small bowel resection, diabetic splanchnic neuropathy, lymphosarcoma, scleroderma and other conditions are associated with the secondary form of malabsorption. The occurrence of prolonged malabsorption following vascular occlusion of the mesenteric arteries despite surgical correction of the occlusion has been reported.[2] Still another interesting instance of malabsorption has been associated with metastatic carcinoid of the bowel.[3]

The role of the radiologist in contributing to the identification of these conditions may be by roentgenologic methods and studies employing radioactive labelled substances.

Golden[4] recently described the technical factors of the roentgenologic examination of the small bowel and he stressed the need for care and attention to detail in performing the study. The mucosal pattern, motility and other morphologic changes of the small bowel may suggest the presence of a malabsorptive state. A preliminary roentgenogram of the abdomen should always be made and it is recommended that any patient with a history of gross steatorrhea of longer than one year's duration should have a roentgenographic survey of the spine, pelvis and chest for evidence of osseous rarefaction. Furthermore, the presence of pancreatic calcification clearly identifies chronic pancreatitis and may be seen on a preliminary abdominal film,[5] although chronic pancreatitis may be present also without such calcification. Golden described the ice-cold saline barium meal, the two-part barium meal, the small intestine enema and Miller-Abbott tube studies as methods available for examination of the small bowel. The ice-cold saline meal is designed primarily as a rapid procedure but may be objectionable because of abnormal

[1] Kabler, J. D., Atwood, W. H., Jr., and Schilling, R. F. Serum turbidity following a fat meal as a test of malabsorption. *J. Lab. & Clin Med.*, 1959, *54*, 427–433.

[2] Joske, R. A., Shamma'a, M. H., and Drummey, G. D. Intestinal malabsorption following temporary occlusion of the superior mesenteric artery. *Am. J. Med.*, 1958, *25*, 449–455.

[3] Kowlessar, O. D., Law, D. H., and Sleisenger, M. H. Malabsorption syndrome associated with metastatic carcinoid tumor. *Am. J. Med.*, 1959, *27*, 673–677.

[4] Golden, R. Technical factors in the roentgen examination of the small intestine. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 965–972.

[5] Gambill, E. E., and Pugh, D. G. Pancreatic calcification; study of clinical and roentgenologic data on thirty-nine cases. *Arch. Int. Med.*, 1948, *81*, 301–315.

stimulus to the gut. As Golden states, "the objective of the procedure is not speed itself but accurate observation." The small intestine enema requires the passage of a tube, as does the Miller-Abbott tube study. The small intestine study, with proper attention to detail, appropriate spot film techniques and high voltage techniques if desired, should disclose abnormalities in a high percentage of cases.

Heretofore, the roentgenologic features of dilatation, segmentation, hypersecretion, and changes in motility and in the mucosal markings have been referred to as a deficiency pattern, disordered motor function, segmentation or irritation pattern, or as being due to enteropathy in deficiency states. Marshak, Wolf and Eliasoph[6] have suggested that this condition might better be referred to as the "sprue pattern." Consideration of the factors responsible for the production of these findings in the small bowel is beyond the scope of this discussion. Dilatation, segmentation and hypersecretion are the most commonly observed changes in the roentgenographic appearance of the small bowel in the presence of malabsorption, and those diseases of malabsorption most commonly associated with these findings are sprue, Whipple's disease and lymphosarcoma.[6]

Diseases which may evidence similar findings but without associated malabsorption are hookworm disease, hyperthyroidism, allergy, emotional disturbances, chronic ulcerative colitis, chronic idiopathic adult tetany, pancreatic insufficiency, icterus and tuberculosis. It is interesting that Golden[7] observed such changes in the small bowel of normal newborns, thereby suggesting damage to the intramural nervous system as an etiologic factor in producing these alterations.

It should be emphasized that these roentgen findings of malabsorption as seen in the small bowel are only indicative of disease as stated and are not specific of a given condition. It must also be remembered that changes seen may depend in part upon the method of examination and materials used, and therefore a thorough knowledge of the technique employed is necessary for accurate interpretation. The flocculation pattern has been perhaps the subject of greatest debate. Paterson and Baker[8] have classified the changes characterizing it into those of clumping, spiculation, granulation and milkiness. It seems unlikely, however, that such fine differentiation will long endure the test of practicality.

The other group of procedures employed by the radiologist in evaluating the malabsorption syndrome utilizes radioactive labelled substances. In the malabsorptive state all of the nutrients are involved and the most common of absorptive defects concerns fat, producing steatorrhea. As Volwiler[9] states, the three best methods of recognizing excessive fat loss are (1) the odor and gross appearance of the stool; (2) microscopic staining for neutral fats and soaps; and (3) intake-excretion studies, the latter being of most value in detecting mild steatorrhea and in quantitating the degree of fat loss. Newer methods which have been developed in this regard involve the determination of absorption of the I^{131} tagged-fat substances, fecal radioiron excretion and radioactive cobalt labelled vitamin B_{12} absorption. The procedure introduced by Ruffin *et al.*[10] employing I^{131} labelled glycerol trioleate has undergone numerous technical modifications which render it easier to perform and which yields better correlation with the presence or absence of steatorrhea. These criteria must be considered in the evaluation of any oral ab-

[6] Marshak, R. H., Wolf, B. S., and Eliasoph, J. The roentgen findings in the malabsorption syndrome. In: The Malabsorption Syndrome. Edited by D. Adlersberg. Grune & Stratton, New York, 1957, pp. 188–205.

[7] Golden, R. Abnormalities of small intestine in nutritional disturbances; some observations on their physiologic basis (Carman Lecture). *Radiology*, 1941, *36*, 262–286.

[8] Paterson, D. E., and Baker, S. J. The radiological investigation of the small intestine in tropical idiopathic malabsorption. *J. Fac. Radiologists*, 1958, *9*, 183–194.

[9] Volwiler, W. Gastrointestinal malabsorptive syndromes. *Am. J. Med.*, 1957, *23*, 250–268.

[10] Ruffin, J. M., Shingleton, W. W., Baylin, G. J., Hymans, J. C., Isley, J. K., Sanders, A. P., and Sohmer, M. F., Jr. I^{131}-labeled fat in the study of intestinal absorption. *New England J. Med.*, 1956, *255*, 594–597.

sorption test for the investigation of the malabsorption state.

Triolein requires the presence of pancreatic enzymes for absorption, and, therefore, affords information as to pancreatic sufficiency as well as to the absorptive state of the small bowel. As a companion test the absorption of I^{131} labelled oleic acid requiring only absorptive integrity enables a differentiation between primary and secondary malabsorption states to some extent. Brown, Glasser and Kurt[11] have also utilized these tests to evaluate the efficacy of treatment.

The absorption of radiocobalt-labelled cyanocobalamin as described by Schilling[12] is still another method by which these conditions can be studied. Best and his coworkers[13] have determined that the percentage of orally administered cobalt excreted in the urine in sprue is 1–2 per cent as compared to 0.1–2 per cent in the presence of pernicious anemia and 9–14 per cent in normal controls. After the administration of intrinsic factor and during repeated study, values rise to 12–19 per cent in pernicious anemia and 2.5–3.5 per cent in the presence of sprue. Of interest in this regard is a recent report by Doscherholmen and Hagen[14] which indicates that some storage of vitamin B_{12} in the intestinal mucosa may influence temporarily the results of tests employing small quantities of vitamin B_{12}. This may throw new light on the reliability of this procedure.

I^{131} labelled protein such as casein and iodinated human serum albumin have also been used on a more limited basis. The serum turbidity test and others will require further investigation. The *in vivo* recognition of the malabsorption state, as verified by jejunal biopsy performed with the Shiner tube or Crosby capsule, has definitely aided the identification of these various conditions.

The development of radioisotope methods of study along with a better understanding of the roentgen appearance of the small bowel and its mucosal patterns thus constitutes a marked progress in the clarification of the malabsorption syndrome. A considerable challenge still exists in this regard and, in fact, is receiving timely impetus. Correlation of the reliability of the various methods of study as well as simplification of the many, often too cumbersome, techniques becomes increasingly important as the ramifications of the malabsorption syndrome become more fully recognized.

K. L. Krabbenhoft, M.D.

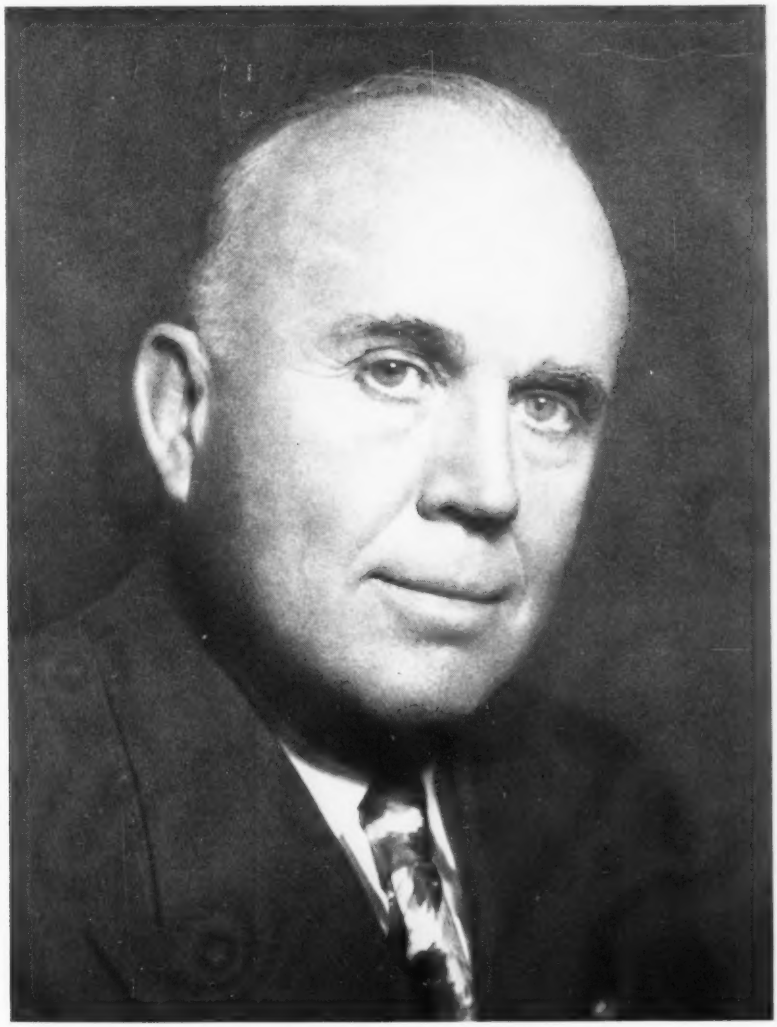
Harper Hospital
Detroit 1, Michigan

[11] Brown, C. H., Glasser, O., and Kurt, E. J. Radioactive fat absorption and clinical response of sprue to steroid therapy. *Gastroenterology*, 1959, *36*, 760–769.

[12] Schilling, R. F. Intrinsic factor studies. II. The effect of gastric juice on the urinary excretion of radioactivity after the oral administration of radioactive vitamin B_{12}. *J. Lab. & Clin. Med.*, 1953, *42*, 860–866.

[13] Best, W. R., White, W. F., Louis, J., and Limarzi, L. R. Experiences with the Schilling test using Co^{60} labeled vitamin B_{12} in pernicious anemia, sprue, and other conditions. *J. Lab. & Clin. Med.*, 1954, *44*, 767–768.

[14] Doscherholmen, A., and Hagen, P. S. Delay of absorption of radiolabeled cyanocobalamin in the intestinal wall in the presence of intrinsic factor. *J. Lab. & Clin. Med.*, 1959, *54*, 434–439.



WILLIAM E. COSTOLOW
1892-1959

SOME men live detached lives; others live to the fullest extent. So it was with "Bill" Costolow, who died November 22, 1959, at the age of sixty-seven years. He was interested in organized medicine, but he did not neglect the social side of life.

Following the completion of his medical education at the University of Pennsylvania, where he graduated in 1916, he spent a year with John Clark at the Lankanau Hospital. It was then he decided to make his professional career one in gynecology, paying particular attention to radium therapy. He also spent two years at the Mayo Clinic, with Bowing, from 1919 to 1921. About this time Al Soiland was looking for an associate in Los Angeles, so Bill Costolow followed Horace Greeley's advice, "Go West, young man." A short time later the writer came on the scene and the firm of Soiland, Costolow and Meland was founded, practicing under the name of

the Los Angeles Tumor Institute. This association continued after the death of Doctor Soiland, until the past year; since then some of the younger men of the group have been carrying on.

Doctor Costolow held a teaching position as Clinical Professor of Therapeutic Radiology at the University of Southern California for many years. He was a member of the Radiological Society of North America, President of the American Radium Society in 1943; and President of the Los Angeles County Medical Association in 1950. He was on the staffs of the California Hospital, the St. Vincent's Hospital, and the Santa Fe Hospital. He was a Trustee of the Albert Soiland Cancer Foundation.

Doctor Costolow was a member and organizer of the Beach Club in Santa Monica, a member of the University Club of Los Angeles, Past Commander of the Albert Soiland American Legion Post, and a Shriner (member of Al Malaikah Temple in Los Angeles).

He served in the Naval Reserve during the two World Wars. In the first war he spent his time on the East Coast, and in World War II he was in the Pacific Theater, being the Senior Medical Officer of the U.S.S. Pinkney. He was retired with the rank of Captain in 1952.

Since Doctor Costolow's return from the second World War, his health has been failing gradually, resulting in less and less activity in medical fields. Doctor Costolow is remembered by his associates, not only as a man but as a physician who acquired a deserved recognition in the field of cancer and its treatment by means of radiation.

Orville N. Meland, M.D.

SOCIETY PROCEEDINGS AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Ambassador Hotel, Atlantic City, N. J., Sept. 27–30, 1960.

American Radium Society
Secretary, Dr. Charles G. Stetson, Englewood Hospital, 350 Engle Street, Englewood, N. J. Annual meeting: Broadmoor Hotel, Colorado Springs, Colo., May 11–14, 1961.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: December 4–9, 1960, Cincinnati, Ohio.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 1961.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: June 13–17, 1960, Miami Beach, Fla.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building. Rochester, Minn. The spring 1960 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, June 6–10; deadline for filing applications was January 1, 1960. The Fall 1960 examination will be held in Cincinnati, December 10 through 13. The deadline for filing applications is July 1, 1960. A Special Examination in Nuclear Medicine (for diplomates in Radiology or Therapeutic Radiology) will be offered, provided there are sufficient applications.

Tenth International Congress of Radiology
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

Seventh Inter-American Congress of Radiology
Secretary, Dr. Laura Fariñas, Havana, Cuba. *Counselor for the United States*, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in São Paulo, Brazil, September 3–10, 1961.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

Arizona Radiological Society
Secretary, Dr. R. Lee Foster, 1313 N. Second St., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. J. B. Scruggs, Arkansas Baptist Hospital, Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. Paul Riemenschneider, Medical College State University of New York, Syracuse, N. Y. Annual meeting to be announced.

Atlanta Radiological Society
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St. Philadelphia, Pa.

Brooklyn Radiological Society
Secretary, Dr. Isadore Katz, 450 Clarkson Ave., Brooklyn 3, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Alfred Dobrak, 710 Leydecker Rd., Buffalo 24, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Radiological Society
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. William B. Schwartz, 1500 W. Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. George B. Cahill, 802 Burns Ave., Flossmoor, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary, Dr. Norman E. Berman, 14404 S. Park Blvd., Shaker Hgts. 20, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

Colorado Radiological Society
Secretary, Dr. Bertram L. Pear, 3705 East Colfax Ave., Denver 6, Colo. Meets third Friday of each month at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. Paul J. Kingston, 114 Woodland St., Hartford, Conn. Meets first Friday in February and April.

Dallas-Fort Worth Radiological Club
Secretary, Dr. W. H. Neil, 1217 W. Cannon St., Fort Worth, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

East Tennessee Radiological Society
Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Building, Knoxville, Tenn. Meets in January and September.

Eastern Radiological Society
Secretary, Dr. John D. Osmond, Jr., Euclid-Glenville Hospital, Cleveland 19, Ohio. Meets at Mid Pines Club, Southern Pines, N. C., March 6–10, 1960.

Florida Radiological Society
Secretary, Dr. John P. Ferrell, 166 4th Ave., St. Petersburg, Fla. Meets twice annually, in the spring with the annual State Society Meeting, and in the fall.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1501 Jungle, St. Petersburg, Fla.

Georgia Radiological Society
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. Donald H. Altman, 2751 Coral Way, Miami, Fla. Meets monthly third Wednesday at 8 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Harvey A. Humphrey, 462 N. Taylor, St. Louis 8, Mo.

Houston Radiological Society
Secretary, Dr. Edward B. Singleton, 6621 Fannin St., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. Charles R. McWilliams, Magic Valley Memorial Hospital, Twin Falls, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in spring and fall.

Indiana Roentgen Society
Secretary, Dr. David E. Wheeler, 1500 North Ritter, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Lewis G. Allen, 807 Huron Bldg., Kansas City, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Robert H. Akers, V. A. Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. C. P. Naidorf, 411 Parkside Ave. Brooklyn 26, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Denis C. Adler, 2010 Wilshire Blvd., Los Angeles 57, Calif. Meets second Wednesday of month in September, November, March, April and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Edward C. Porter, 489 State Street, Bangor, Maine. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. Hollis H. Halford, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. S. F. Johnson, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Bernard T. Hickman, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. J. K. Boughn, 35 11th Ave., Helena, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Jerome Zwanger, 126 Hicksville Rd., Massapequa, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. Ronald E. Waggener, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. John E. Gary, 1180 Beacon St., Brookline 46, Mass. Meets third Friday of each month, October through May, at The Longwood Towers, Brookline, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. William B. Seaman, 622 W. 168th St., New York, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. Owen Doyle, 906 Dover Road, Greensboro, North Carolina. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. R. F. Raasch, Post Office Box 990, Dickinson, North Dakota. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Paul A. Mori, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Paul D. Meyer, 125 S. Grant Ave., Columbus, Ohio. Annual meeting to be announced.

Oklahoma State Radiological Society
Secretary, Dr. J. Murphree, 617 E. Brookfield, Ponca City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Barbara J. Radmore, 1162 Willamette, Eugene, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. Arch Colbrunn, 19 Royal Oak Drive, Roseburg, Ore. Annual meeting: Vancouver, British Columbia, May, 1960.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Walter P. Bitner, 234 State St., Harrisburg, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Robert B. Funch, Department of Radiology, Germantown Hospital, Philadelphia 44, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Charles N. Chasler, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenley Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. Stanley J. Lucas, 417 Provident Bank, Cincinnati 2, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Louis L. Buzaid, The Queen's Hospital, Box 861, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. Gerhard W. Schottman, 4400 J. C. Nichols Pkwy., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Robyn Hardy, 4324 Magnolia St., New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Austin J. Tidaback, 912 Prospect Ave., Plainfield, N. J. Meets at Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Robert H. Greenlaw, 188 Irvington Rd., Rochester 20, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 11–13, 1960.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at Grison's Steak House.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Ralph J. Littwin, Bristol Hospital, Bristol, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1835 Eye St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary. Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. Annual meeting: Estes Park, Colorado, June 22–25, 1960.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. Wayne Reeser, 804 Burroughs St., Conway, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Gordon Black, Suite 2-A, El Paso Medical Center, 1501 Arizona, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. James J. Range, P.O. Box 324, Johnson City, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Next meeting January 20 and 21, 1961, Texas Hotel, Fort Worth, Texas.

Tri-State Radiological Society
Secretary, Dr. James R. Mathews, 118 S. E. First St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Frank A. Kearney, II, 110 S. Curry St., Phoebus, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Wayne A. Chesledon, 306 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. James C. Montieth, 170 Maple Avenue, White Plains, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

X-Ray Study Club of San Francisco
Secretary, Dr. John H. Heald, 450 Sutter St., San Francisco 8, Calif. Meets monthly, third Thursday at 7:30 P.M., Children's Hospital, September through June.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2-37 zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Honorary Secretary, Dr. John Blewett, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting: June 10–11, 1960.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Louis Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

O Colégio Brasileiro de Radiologia
Secretary-General, Dr. Walter Bomfins Pontes. Seat: Rua Major Quedinho, 99-2°. Andar, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Juan Sabbagh Dacla, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Annual meeting: May 11–14, 1960.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The next list of Meetings of Radiological Societies will be published in the July issue of the Journal.

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

At a meeting held at The Caribe Hilton Hotel in San Juan, Puerto Rico, on March 17–19, 1960, the following officers were elected: President, Jesshill Love, Indianapolis, Ind.; President-Elect, Robert L. Brown, Atlanta, Ga.; 1st Vice-President, Harold W. Dargeon, New York, N. Y.; 2nd Vice-President, Victor A. Marcial, Puerto Rico; Secretary, Charles G. Stetson, Englewood, N. J., and Treasurer, Gilbert H. Fletcher, Houston, Texas.

The Forty-third Annual Meeting will be held in Colorado Springs, Colorado, May 11–14, 1961.

THE ROCKY MOUNTAIN RADIOLOGIC SOCIETY

The next annual meeting of the Rocky Mountain Radiologic Society will be held in the new Denver Hilton Hotel in Denver, Colorado, August 11–13, 1960. Among the guest speakers will be Doctors Ted F. Leigh, C. Richard Perryman, Sidney F. Thomas, Benjamin Felson, and David M. Gould.

THIRD SYMPOSIUM ON CINEFLUOROGRAPHY

Plans are now underway for the Third Symposium on Cinefluorography to be held at the University of Rochester School of Medicine and Dentistry in the Spring of 1961.

A meeting on cinefluorography is not planned for the Fall of 1960. Further announcements concerning the symposium will be published later.

INTERNATIONAL ATOMIC ENERGY AGENCY ARRANGES CONFERENCE

A major scientific conference to be arranged by the International Atomic Energy Agency in 1960 will be held in Copenhagen, September 6–17.

The conference will deal with the use of radioisotopes in the physical sciences and industry and it is expected that more than 500 scientists from the Agency's seventy Member States will attend.

The Danish Government has invited the Agency to hold this conference in its capital city and promised to arrange for the necessary facilities. The Director General has accepted the Danish offer and expressed his satisfaction at this generous support by Denmark of the Agency's scientific conference program.

It is expected that the results of recent research work will be submitted and discussed and the rapid progress that has taken place recently in this field will thus be made available to all countries.

The last major scientific conference on radioisotopes was held in Paris in September, 1957 and was organized by Unesco. Unesco is cooperating with the International Agency in the preparation for the Copenhagen conference. Uses of radioisotopes was also the subject of a considerable number of papers submitted to the second United Nations Conference on the peaceful uses of atomic energy held in Geneva in September, 1958.

ATOMIC ENERGY COMMISSION SPECIAL FELLOWSHIPS

Two hundred and thirty-one graduate students from thirty-nine states, the District of Columbia, and Puerto Rico have been awarded Atomic Energy Commission Special Fellowships in Nuclear Science and Engineering.

Administered for the Commission by the Oak Ridge Institute of Nuclear Studies, the fellowships are designed to aid and encourage promising students who are studying for master's or doctor's degrees in nuclear fields. Fellowship appointments are made for twelve months, and may be used for study at one of 49 designated universities throughout the country.

RADIOACTIVE COURSE AT THE UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL

An intensive didactic, laboratory and clinical postgraduate course in the diagnostic and therapeutic uses of radioiodine is announced.

The course is intended to qualify the enrollee for Atomic Energy Commission licensure. Enrollment is limited to ten in a course to be conducted from June 20 through June 25, 1960. Additional information may be obtained from Frederick J. Bonte, M.D., Professor and Chairman, Department of Radiology or John S. Chapman, M.D., Assistant Dean, University of Texas Southwestern Medical School, 5323 Harry Hines Boulevard, Dallas, Texas.

NINTH ANNUAL IRA I KAPLAN LECTURE

The ninth annual Ira I Kaplan Lecture will be held Thursday, May 26, 1960, 5:30 P.M. in the main auditorium, Alumni Hall, New York University School of Medicine, 550 First Avenue at 31st Street, New York, New York. The lecturer will be Dr. Sidney Farber, Professor of Pathology, Harvard Medical School, at the Children's Hospital, and Director of Research, Children's Cancer Research Foundation. Dr. Farber will speak on "Biological and Therapeutic Considerations of Cancer in Early Life."

SEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY MEETING DATE ANNOUNCED

The Seventh Inter-American Congress of Radiology is to be held in São Paulo, Brazil, on September 3–10, 1961. The President of the Congress will be Dr. Jose M. Cabello-Campos and the Secretary-General will be Dr. Walter Bomfim-Pontes, Rua Cesario Motta, No. 112, São Paulo, Brazil.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Blood and Lymph System

Hoeffken, W. Die Angiokardiographie mit Kohlendioxyd. (Angiocardiography with carbon dioxide.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, July, 1959, *91*, 1–13. (From: Röntgen-Abteilung der Med. Univ.-Poliklinik, Bürgerhospital, Köln, Germany.)

Angiocardiography with iodine contrast solutions has proved to be a valuable diagnostic procedure. Its disadvantages, however, are occasional iodine reactions and the fact that details of valvular structures, function and wall thickness are obscured by the opaque material. These drawbacks can partly be overcome by the use of carbon dioxide gas instead of an iodine solution.

The gas is rather innocuous as it can easily be absorbed by the venous blood in quantities up to thirty times larger than air. A dose of 100 cc. usually is well tolerated and can safely be repeated after an interval of ten minutes. Side effects are minimal; the electrocardiogram may show only an occasional extrasystole. A left lateral recumbent position is advised as a precautionary measure to prevent or alleviate symptoms and signs of gas embolism. A right to left shunt forms a contraindication although in experimental animals the gas is well tolerated when injected into the left ventricle and the peripheral arteries.

Fluoroscopically, the injected gas is well seen for a period of fifteen seconds with residual bubbles remaining up to ten minutes. Spot roentgenograms will show the gas in the superior vena cava, right atrium and ventricle, pulmonary valve and trunk, and in the inferior vena cava. The tricuspid valve and papillary muscles are only seen in tangential projection. Wall thickness of the right heart chambers with or without hypertrophy can easily be recognized.

The prone position in addition to the supine is advantageous for showing the posterior wall of the vena cava. In the left lateral recumbent position the right atrium, being at the highest point, contains a horizontal blood level and will remain filled for a prolonged period of time. The right lateral recumbent position is best for showing the pulmonary trunk and for excluding stenosis of the pulmonary valve. Only small amounts of gas are advised in this position because of the danger of blocking the lesser circulation. Therefore, after completion of spot filming, the patient has to be quickly turned to the left side so that the gas may become trapped in the right atrium and ventricle instead of going to the lungs.

For serial roentgenography best results can be obtained with an Odelca camera. Cineroentgenography with an image intensifier is advantageous for illustrating function.

The method is most helpful for determining the size and wall thickness of the right heart chambers and for possible intrinsic and extrinsic changes of the vena cava. It also is of value for recognizing pericardial processes, thrombi, tumors, aneurysms, and congenital as well as acquired lesions of the tricuspid and pulmonary valves. The article is well illustrated. —*Ernest Kraft, M.D.*

Porstmann, W., and Geibler W. Die retrograde Katheterisierung des linken Ventrikels von der Arteria femoralis und der Arteria carotis communis dextra: Zwei sich ergänzende Methoden, ihre Indikationen und Ergebnisse. (Retrograde catheterization of the left ventricle from the femoral and right common carotid arteries: two methods supplementing each other, their indications and results.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, July, 1959, *91*, 14–24. (From: Institut für Röntgenologie der Charité und der I. Medizinischen Universitätsklinik der Charité, Berlin, Germany.)

Cardioangiography by direct puncture of the left ventricle has generally been abandoned because it is a hazardous procedure. Therefore, it has more recently been replaced by retrograde catheterization which is simpler, less harmful and more informative.

The authors have used the new approach in 76 cases with 25 carotid and 51 femoral artery catheterizations. Percutaneous femoral artery catheterization is preferable and succeeds in most cases; but in severe stenosis of the aortic valve it may become necessary to select the right common carotid artery instead. This approach represents a short and straight downward path so that the catheter can be more easily manipulated into the left ventricle. Thus, only 4 failures have been encountered in 25 cases compared to 15 failures in 51 cases in which the percutaneous femoral artery method was used.

The examination consists of the following two parts: (1) intracardiac measurement of pressure and (2) contrast visualization. The second part can be omitted when pressure readings above and below the aortic valve are characteristic of a stenosis. In subvalvular aortic stenosis, however, contrast visualization is of value to rule out secondary incompetence of the adjacent mitral valve leaflet.

A left to right shunt can be recognized, but the method is only partly useful in mitral insufficiency, which is frequently caused by extrasystoles. These always occur when the tip of the catheter comes in contact with the wall of the heart chamber. In aortic insufficiency the method is more reliable, as contrast material is injected into the ascending aorta where extrasystoles cannot be induced. In combined lesions of the aortic valve the stenotic phase can again be determined by the pressure gradient. When the

catheter cannot be passed into the left ventricle, even with the carotid approach, abnormally low pressure readings in the aorta may explain the cause of resistance. In such rare instances an injection into the ascending aorta is recommended.

Numerous roentgenographic reproductions and curves accompany this interesting article.—*Ernest Kraft, M.D.*

Schmidt, H. W., and Deupmann, F. J. Diagnostische Möglichkeiten und Grenzen der Angiographie mit Sauerstoff und Kohlendioxyd. (Diagnostic potentialities and limitations of angiography with oxygen and carbon dioxide.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, July, 1959, *91*, 25–34. (From: I. Medizinische Klinik der Medizinischen Akademie, Düsseldorf, Germany.)

Angiography of peripheral arteries has been used with good results for many years. The method, however, is contraindicated in patients with iodine sensitivity. Another disadvantage is the fact that details of pathologic changes may be obscured by the opaque material. Gas arteriography, which is well tolerated, shows finest details and fades gradually without causing harm. When the femoral artery on the affected side cannot be palpated, injection of the contralateral artery may succeed in getting gas in retrograde fashion via the aortic bifurcation to the desired location.

At first percutaneous injection of carbon dioxide gas was tried. Pure oxygen, however, has proved superior because of a much slower rate of absorption. Therefore, filming can be done at a rather leisurely rate, and no specific equipment for serial roentgenography is necessary. Initial exposures are made after an injection of 40 cc. and final filming takes place following an additional dose of 40 cc.

In a series of 55 cases, the procedure has caused no side effects or arterial spasm. The gas, however, is too quickly absorbed to be of diagnostic value in the popliteal and peripheral arteries. In the upper extremities the gas cannot be used as it may easily escape into the carotid arteries toward the brain.

With the gas, formerly unrecognized vascular calcifications appear clearly. Arterial occlusion gives a very striking picture, with gas visualization of the peripheral segment. In fresh embolism, however, narrowing of the vascular lumen cannot be recognized if the clot does not cling to the wall.

Vascular spasm has not been observed in this series of 55 cases, and it is the impression of the authors that, in regular contrast arteriograms, spasm is frequently simulated by incomplete mixture of blood and opaque material.—*Ernest Kraft, M.D.*

Abrams, Herbert L. An approach to biplane cineangiocardiography. 1. Background and objectives. *Radiology*, May, 1959, *72*, 735–740. (Address: Stanford University School of Medicine, San Francisco 15, Calif.)

The study of cardiac pathology using injected opaque media in conjunction with various roentgenologic techniques has involved extensive and varied experimentation. Roentgen cinematography is the most recent addition to this field of endeavor and its application to the diagnosis of heart disease has proved extremely valuable. The present article deals with the many problems inherent in its application and demonstrates how some of these problems may be or have been overcome. The historical background of the development of angiocardiographic techniques is presented and a fine bibliography is included if additional information is desired.

The first angiocardiographic studies performed by Castellanos and co-workers employed a single film exposed when the opaque medium was thought to be in the right cardiac chambers. This and other similar techniques proved to be unsatisfactory in many cases, and the need for multiple exposures at a rapid rate became apparent. This gave rise to the construction of various high speed cassette changers, roll film and cut film magazine cameras. The cine technique is ideally suited for this purpose and has satisfied most of the needs in this direction.

The advent of image intensification solved, to a great degree, the problem of obtaining adequate light for film exposure without excessive radiation to the patient and staff. In addition to this, exposure has been synchronized with the camera shutters so that patient irradiation occurs only when the film is being exposed.

One of the greatest problems in cinefluorography has been the relatively poor film definition obtained compared to the fine detail of the full size roentgenogram taken during conventional angiocardiography. The development of faster lenses, more sensitive and fine grain film, superior processing solutions and better projection apparatus has obviated some of these disadvantages. Also in this regard, the development of electronic switching systems has made possible ultra-short exposure times in the range of milliseconds. Such exposures should theoretically permit "stopping of cardiac motion" with consequent diminution of motion blurring. Another problem encountered was the overlapping of cardiac chambers during the passage of the opaque medium which obscured many important intra- and extracardiac details. Thus, a right to left shunt at the atrial level may be apparent in one projection, but in the same projection the outflow tract of the right ventricle and pulmonic valve may be obscured. To overcome this, single plane techniques have been utilized in such a way that two dimensional or biplane studies can be obtained. Two single plane units can be coupled at right angles to one another and synchronized in such a way that exposures can be made simultaneously in both planes.

Fixing the precise moment of the cardiac cycle at which an exposure is made would be useful in providing multiphase study and analysis of anatomic, physiologic and electric events occurring in the heart. To this end various apparatus to record right ventricular pressure, carotid pressure, electrocardiogram and phonocardiogram, all taken concurrently with the cinefluorographic study, have been utilized. The quantity and rate of injection of the opaque medium required standardization and control. For this purpose, both manual and remotely controlled automatic pressure injectors have been devised. Other aspects such as film viewing apparatus, screen size, and patient size in relation to successful cineangiocardiography are discussed. Improvements in this field are expected to follow continued experimentation.—*Z. Petrany, M.D.*

Abrams, Herbert L. An approach to biplane cineangiocardiography. 2. Equipment and procedure. *Radiology*, May, 1959, *72*, 741–750. (Address: Stanford University School of Medicine, San Francisco 15, Calif.)

The author discusses the procedure and installation employed in biplane cineangiocardiography. For those interested in the technical details, the article should be read in its entirety. Eleven-inch image intensifiers specifically designed for cinefluorography are used and each intensifier is equipped with two monocular viewers enabling two persons to view the image simultaneously. The vertical intensifier is mounted in a cave beneath the floor level and is fixed in position in relation to the specially constructed adjustable table. The horizontal intensifier rests on a mobile base and may be moved away from the table when not in use. A 35 mm. motion picture camera is an integral part of the image intensifier system and film speeds of 6, 12, 16, 24, 32, and 48 frames per second may be obtained by the proper setting of the instrument. Two exposure meters are mounted adjacent to the control panel and along with photo-electric cells attached to the camera allow the technique to be determined empirically with reasonable accuracy. The Dynapulse unit, generator, and roentgen-ray tubes employed are described.

Fluoroscopy, with both 11 inch image intensifiers and the 5 inch intensifier, is mediated by the control panel, although the actual fluoroscopic switches are controllable by the operator at the table if desired. When cinefluorography is utilized the switch is easily made through the control panel, and separate knobs determine the milliamperage on the horizontal and vertical tubes. The kilovoltage is preset and must be the same for both planes although the time and milliamperage may be varied independently with the Dynapulse unit. A synchronizing device is incorporated in the unit to synchronize the 2 planes not only to determine the relationship of the exposure of one plane to the other but also so that scatter may be diminished.

In actual operation, the patient is placed on the table with the head toward the 5 inch image intensifier which is used for fluoroscopy only in order to check catheter placement. With the chest overlying the 11 inch intensifier, the field size and patient's position are studied and changes made as required. The controls are set individually for the horizontal and vertical intensifiers and the catheter is placed fluoroscopically. The patient is then moved into cine position and the procedure is started. Throughout the examination, electrocardiographic and exposure records are taken. The author stresses the need for an operating check list in order to minimize the possibility of human error.—*John S. Alexander, M.D.*

Urschel, Harold C., Jr., and Roth, Earl J. Electronically controlled coronary arteriography. *Ann. Surg.*, Aug., 1959, *150*, 275–289. (From: Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.)

An experimental method of opacification of the coronary arteries by a single injection of a small amount of contrast material is described by the authors. The animals used were dogs. A catheter (#12) was inserted into the coronary sinus through the right carotid artery. The contrast medium was injected by an electronically controlled apparatus and the roentgen-ray exposure made and timed by the same apparatus. By trial and error it was determined that the best coronary filling was obtained when the medium was injected just before mid-diastole and the roentgen-ray exposure made at the end of the same diastole. When this procedure was followed, maximum opacification of the coronary arteries was obtained with the injection of only 1 cc. of medium in one-tenth of a second. Fifty per cent miokon was found to be the best concentration to allow adequate flow in the coronary arteries and satisfactory opacification of these vessels.

Using this method, a single injection and a single roentgen-ray exposure produced good results.

The electronically controlled apparatus is described in detail and illustrated with diagrams. The method has not been tested adequately on human subjects at present.—*Frank M. Windrow, M.D.*

Semisch, Robert. Diagnostische Möglichkeiten der selektiven Lungenangiographie. (Diagnostic possibilities of selective pulmonary angiography.) *Thoraxchirurgie*, June, 1959, *6*, 551–564. (From: Chirurgische Universitätsklinik, Jena, East Germany.)

In a normal pulmonary angiogram, three characteristic phases can usually be distinguished: the

precapillary arterial phase, the phase of capillary passage, and the phase of venous drainage. The vessel outline is dense throughout, with a moderately curved or tortuous course. The angle of ramification is less than 60 degrees and the tapering of the vessels is continuous to the periphery, reaching a diameter of 0.15 mm. Selective pulmonary angiography has proved to be eminently suitable for clarifying morphologic deviousness in the pulmonary blood flow. The author describes its application to a variety of diagnostic problems.

Direct morphologic deviation of the pulmonary artery, for instance in bronchogenic carcinoma, is not rare and is quite significant. It may indicate invasion or encircling of a main pulmonary vessel by a tumor, which implies inoperability.

Pulmonary angiography is able to differentiate a reversible from an irreversible atelectasis. Bolt and Rink set as a criterion the visualization of a capillary filling phase to determine whether the terminal pulmonary segment is still able to function.

This technique used in cases of pulmonary tuberculosis reveals, in addition to grotesque morphologic distortions of the great vessels and a high degree of rarefaction of the vessels, the influence of localized, deficient alveolar aeration with pronounced secondary strangulation of the blood flow. In many instances, only the pulmonary angiogram is able to demonstrate the actual extent of a tuberculous pulmonary process, especially when there is extensive pleural thickening.

Determination of the degree of chronic pulmonary emphysema is of vital importance prior to a lung operation. The author has devised a special technique which he has named "terminal pulmonary angiography." A detailed account of this procedure, as well as the physiologic and pathophysiologic inferences, will be published. The obstructive form of pulmonary emphysema destroys the alveolar capillary structures and, in time, the collateral circulation is reduced, increasing the load on the right heart. In this situation, development of cor pulmonale or acute failure of the right heart is unavoidable following resection of the lung. The general criteria for chronic pulmonary emphysema are: stretching of the arteries, increase of the branching angle, and marked reduction of the diameter of the vessels. Four grades of emphysema are recognized, according to Rossier. When pneumonectomy is considered, the case should not be more advanced than Grade II; a case with Grade III may undergo only a lobectomy; and all thoracic surgical interventions are absolutely contraindicated in Grade IV.—*S. Silvera, M.D.*

CARTWRIGHT, ROBERT S., and BAUERSFIELD, S. RICHARD. Thoracic aortography in infants and children. *Ann. Surg.*, Aug., 1959, *150*, 266–274. (From: Department of Cardiology, Children's Hospital of Pittsburgh and the Division of Thoracic Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pa.)

The most common indication for thoracic aortography is a left to right shunt in which a patent ductus arteriosus is suspected. The authors performed thoracic aortography on 92 patients with 83 successful aortograms. Sixty-nine of these cases were in the patent ductus category. Twelve patients were thought to have had simple coarctation. If coarctation is well established by clinical methods, then aortography is performed only on female patients. Unusual types of coarctation are common enouth in young females, and "young female" arteritis may produce findings enough like those of coarctation, to justify the aortography.

The left carotid artery is used and a catheter is inserted 2–4 cm. downward in the common carotid artery after exposing the vessel surgically. Ten cc. to 20 cc. of 35 per cent miokon is injected rapidly. At the same time, the left carotid artery is occluded with a ligature and the right carotid is occluded by the anesthetist to reduce the amount of contrast medium entering the brain. Three deaths occurred. Cannulation of the ascending aorta is avoided because it must be done under fluoroscopy and hemorrhage may occur without the operator's knowledge and occasionally the catheter may enter a coronary sinus. General anesthesia is preferred. Several nonfatal complications were observed. There was 1 Jacksonian convulsion, 1 grand mal convulsion, 2 aspiration pneumonias and 1 bleeding from an unknown source into the bronchial tree.

A definitive diagnosis was made in 34 patients (41 per cent). Only 10 of the patients were over two years old. Filling of the aorta from the sinuses of Valsalva to the diaphragm was obtained in 32 patients. It is stressed that a small volume of only moderately concentrated medium is desirable to minimize cerebral symptoms. Of the last 53 cases of the authors, 52 were successful and there were no deaths.—*Frank M. Windrow, M.D.*

ACTIS-DATO, A., TARQUINI, A., DUGHERA, L., QUAGLIA, C., and WEISZ, R. Morphologisch-dynamische Untersuchung der Aortenklappenfehler mittels retrograder Aortographie. (Dynamic morphologic examination of the aortic valve's defect by means of retrograde aortography.) *Thoraxchirurgie*, June, 1959, *6*, 541–550. (From: Centro di Cardiochirurgia "A. Blalock," Clinica Chirurgica dell'Università di Torino, Torino, Italy.)

The need for an exact evaluation of the anatomic, as well as the functional, condition of the aortic semilunar valve was widely recognized when transventricular or transaortic valvulotomy was first considered. The possibility of surgical treatment of

aortic stenosis, alone or combined with a mitral defect, and of an aortic defect with predominant stenosis, and lesser insufficiency, as well as the necessity of identifying a coexisting aortic defect and persistent ductus arteriosus or an aortic isthmus stenosis, gave rise to ever more refined and exact examination methods, two of which are angiocardiography and retrograde aortography.

Retrograde aortography brings the contrast medium into immediate proximity with the anatomic structures to be examined and is, therefore, more suitable than angiocardiography for determining changes in the diameter of the aorta, morphologic and functional changes in the valve, and the possible existence of backflow of the dye into the left ventricle. Also, it is possible by this method to visualize an existing persistent ductus arteriosus or an aortic isthmus stenosis.

The authors use the following procedure: After infiltration with 1 per cent novocain solution, the right brachial artery is exposed about 6 to 7 cm. proximal to the elbow joint and is incised longitudinally for the passage of a polyethylene tube 2 to 3 mm. in diameter. Under fluoroscopic control, the tube, which contains a flexible radiopaque wire, is threaded through the subclavian artery and the brachiocephalic trunk into the aortic arch. Twenty to 40 cc. of 50 per cent hypaque is injected and the carotid arteries are compressed bilaterally to avoid massive embolism of the brain circulation. With the patient in the right posterior oblique position, a series of roentgenograms are made at the rate of 4 per second in the first two seconds, followed by 2 per second in the next two seconds. The exposures do not exceed 1/50 to 1/25 second through a Bucky diaphragm. They are synchronized photoelectrically with an electrocardiographic apparatus and are made during the mesosystolic period (S-T interval) and mesodiastolic period (at the end of the T wave).

The authors examined 3 normal subjects, 10 patients with aortic stenosis and 9 with aortic insufficiency. Of the 9 with aortic defects, 6 also had mitral stenosis. The ages ranged from seventeen to thirty-eight. All were in satisfactory general condition, without heart or coronary decompensation symptoms. The aortographic findings are as follows:

Normal Cases. Morphologic findings: moderately regular widening of the aortic bulb; regular and sharply defined, curved valve cusps; the ascending aorta uniformly cylindrical in shape. Hemodynamic findings: systolic-diastolic change in the aortic diameter of 3 to 4 mm.; flattening and separation of the internal profile of the individual valve leaflet during systole and crowding of the internal outline during diastole; the valve plane shifting during systole in a cranio-caudal direction and during diastole in a caudo-cranial direction by about 0.3–0.5 cm.

Aortic Stenosis Cases. Morphologic findings: spindle shaped ectasia of the ascending aorta; irregular and serrated edge of the valve leaflet outlines with one of the three valve cusps not visualized due to destruction; calcification, in some instances. Hemodynamic findings: rigidity of the valve cusps and moderate systolic-diastolic shifting of the valve leaflets; no reflux of the dye into the left ventricle; delayed emptying of the aorta.

Aortic Insufficiency Cases. Morphologic findings: extensive ectasia of the ascending aorta; ectasia and deepening of the valve cusps with polycyclic bordering; partial or complete lack of visualization of one valve cusp. Hemodynamic findings: great systolic-diastolic diameter change of the aorta; stronger cranio-caudal shifting of the valves during systole; backflow of the dye into the left ventricle. Where there was coexisting mitral insufficiency, there was reflux of the dye into the left auricle.—*S. Silvera, M.D.*

Fuchs, W. A., Rüttimann, A., and del Buono, M. S. Klinische Indikationen zur Lymphographie. (Clinical indications for lymphography.) *Schweiz. med. Wchnschr.*, July, 1959, *89*, 755–759. (From: Röntgendiagnostisches Zentralinstitut des Kantonsspitals Zürich, Zürich, Switzerland.)

Despite great advances in the last few years, roentgenographic diagnosis of disorders of the lymph vessels and lymph nodes is still in its infancy. However, simplification of the technique and accumulated clinical experience will increase its effectiveness, especially in the early diagnosis of malignant tumor metastases to the lymph nodes. After a short description of the anatomy of the lymph vessels and lymph nodes, as seen roentgenographically, the authors present several selected clinical cases to demonstrate the diagnostic possibilities of lymphography.

The main indications for this examination are edema of unknown origin and tumor infiltration of the pelvic, inguinal and axillary lymph nodes. Of course, edema of cardiac, renal, endocrine or other origin must first be excluded. A deep seated venous thrombosis causing edema can be detected by phlebography; a congenital arteriovenous aneurysm or other arterial disease may be excluded by arteriography. Lymphography has a much broader application in that it will differentiate a primary lymphedema due to congenital changes in the lymph vessels as aplastic, hypoplastic or varicous and it will designate the cause of secondary lymphedema due to mechanical obstruction of the lymph circulation as tumor infiltration, inflammatory vascular changes, injury, hematoma, etc.

Lymphography is especially indicated when tumor infiltration of primary or metastatic origin is clinically suspected in the pelvis, inguinal region or axilla. This applies particularly to cases of carcinoma of the fundus and cervix uteri, prostate, rectum, breast, bladder, and ovary, as well as lymphogranuloma

and lymphosarcoma. It is possible to determine exactly the extent of metastasis of these tumors to the pelvic lymph nodes, although, clinically, it is not suspected.—*S. Silvera, M.D.*

General

Maresh, Marion M. Linear body proportions; a roentgenographic study. *A.M.A. J. Dis. Child.*, July, 1959, *98*, 27–49. (From: The Child Research Council and the University of Colorado School of Medicine, Denver, Colo.)

This study deals with the changes during growth and the variation observed in the linear proportions of body segments in the young adult. By use of the formula, measured bone length/height × 100 = relative bone length, the differences in height of individuals of the same age were negated and it was possible to describe a child as "relatively long-legged" regardless of height at any age. The data were acquired by measurements from seritatim roentgenograms of about 200 persons, of whom less than half had completed their long bone growth.

It was noted that, as the height, sitting height and length of all the long bones increased with growth of the child, the rates of growth were not alike. The long bones became a greater per cent of height from infancy to adolescence, while the relative trunk length became an increasingly smaller per cent of height during the same period. From six months to three years of age, the increase in the relative lengths of the bones of the extremities was most rapid. During growth the changes in relative lengths were less for the humerus and radius than for the femur and tibia.

This study presented evidence that there was a wide range of variation in the comparative relative lengths of the long bones of the extremities and the relative sitting height in the same person and between individuals. There was no correlation between the relative lengths of any long bone and the total height in individuals of the same height. It was also concluded that the segmental proportions in siblings will be alike more often than in nonsiblings.—*Cecil W. Ely, M.D.*

Norman, J. C., and Rizzolo, P. J. Subcutaneous, mediastinal and probable subpericardial emphysema treated with recompression. *New England J. Med.*, Aug. 6, 1959, *261*, 269–272. (From: The United States Naval Submarine Base, New London, Conn.)

A rather interesting case of this syndrome occurring in a healthy young Submarine School candidate is presented. The following symptoms developed after routine compression pressure testing during escape training: a feeling of pressure in the retrosternal space intensified by coughing, sudden motion or attempts at speaking, slight dyspnea and difficulty in phonation.

Physical examination showed minimal injection of the posterior pharynx and subcutaneous emphysema with crepitation to the angles of the jaw, bilaterally. All laboratory tests were normal. A posteroanterior roentgenogram of the chest revealed air in the area of the mediastinum with extension cephalad into the superior retrosternal area and anterior root of the neck. Questionable elevation of the pericardium along the left border of the heart was noted.

Several hours later, subcutaneous emphysema had increased and a definite mediastinal "crunch" synchronous with the cardiac cycle was present. Pain and tenderness of the retromediastinal space were more intense. The patient was transferred to a recompression chamber. At about three atmospheres of pressure the dyspnea subsided and the "crunch" diminished; at four atmospheres the dyspnea was gone, the subcutaneous emphysema had disappeared and the "crunch" was barely audible; at six atmospheres the patient was asymptomatic. He remained in the chamber for nineteen hours and was then transferred to the station hospital where roentgenograms still showed air along the left cardiac border. He was discharged several days later when the chest roentgenogram was negative.

In analyzing the cause of this condition, it was concluded that a minimal upper respiratory infection associated with an overenthusiatic performance of the Valsalva maneuver to equalize pressure in the ears caused an alveolar rupture, with air proceeding along the bronchial adventitial planes or along pulmonary vessels.

Had no recompression chamber been available, the outcome would very likely have been worse with, possibly, surgical intervention being necessary. The recompression method of treatment has only been reported on one other occasion. The authors believe that this case is important because of the increasing number of skin divers and would-be frogmen.—*Louis C. Lippert, M.D.*

Sobin, Sidney S., Frasher, Wallace G., Jacobson, George, and VanEeckhoven Frank A. Nature of adverse reactions to radiopaque agents; preliminary report. *J.A.M.A.*, July 25, 1959, *170*, 1546–1547. (Address: S. S. Sobin, 1200 N. State St., Los Angeles 33, Calif.)

The authors studied the effect of intravenous injection of several of the tri-iodobenzoic acid compounds on the small blood vessels at the corneal-scleral junction in man and rabbits, with a modified binocular dissection microscope.

Following injection of the contrast media for intravenous urography, characteristic changes oc-

curred in these vessels, varying in degree from patient to patient. The changes began one to two minutes after the end of the injection and consisted of marked slowing of the microcirculation and a coarse granular appearance of the red blood cell masses with clear, irregular, intravascular plasma between the masses. There were variable degrees of vasoconstriction noted in both the arterial and venous vessels. At times there was considerable pinching of the red blood column with the formation of sausage-like masses moving slowly down the vessel. Actual stasis did not occur. An over-all decreased vascularity was noted. The intensity of these changes was maximal at four to six minutes after the injection and the vascular bed returned to normal after twelve minutes. The authors had no explanation for the precise cause or nature of these blood vessel changes.

This report is a progressive step in the investigation of the unknown phenomenon that results in sometimes fatal reactions to the intravenous administration of radiopaque agents.—*Everett L. Pirkey, M.D.*

Gil Gayarre, Carlos. Cancer de recto producido por aceite mineral quemado. (Cancer of the rectum caused by burned mineral oil.) *Acta iber. radiol. cancerol.*, July-Sept., 1958, *13*, 271–280.

This case report concerns a male patient who, since the age of nine years, had suffered from rectal polyposis with occasional prolapse through the anus. At the age of sixteen years he went to work for a garage, being assigned to service automobiles, mainly to drain and change the motor oil. At about that time, there occurred an exacerbation and the polyp had to be pushed back through the anus after each defecation. The patient discovered soon that local lubrication will facilitate the reduction of the prolapsed polyp. As a lubricating agent he used the (burned) oil drained from the engines he serviced. Slowly, the condition worsened and, when he reached the age of eighteen years (1957), he was subjected to a biopsy, which demonstrated the presence of adenocarcinoma. Abdominoperineal resection was followed by local recurrence, extension into the sacrum, and metastasis to the inguinal lymph nodes. Telecobalt therapy was then given to the site of recurrence and to the lymph nodes; local examination failed to reveal gross evidence of neoplasia shortly after completion of irradiation (1958).

The case is reported because the author believes that the repeated application of (burned) mineral oil to the rectal polyp was the precipitating factor in the appearance of local malignant degeneration. Nevertheless, he hesitates to call it "occupational cancer" since the repeated manipulations were not directly connected with the patient's work assignment.—*E. R. N. Grigg, M.D.*

RADIATION THERAPY

Stearns, M. W., Jr., Deddish, Michael R., and Quan, Stuart H. Q. Preoperative roentgen therapy for cancer of the rectum. *Surg., Gynec. & Obst.*, Aug., 1959, *109*, 225–229. (From: The Rectum and Colon Service, Memorial Center for Cancer and Allied Diseases, New York, N. Y.)

In the past, many patients with cancer of the rectum have received external roentgen therapy at Memorial Center for Cancer and Allied Diseases, some for palliation and some preoperatively. The authors have evaluated the latter.

Of 1,276 patients operated upon for rectal cancer between 1939 and 1951, 549 received no preoperative roentgen therapy; 727 received such therapy. The treatments were usually given with a 250 kv. unit (1.5 mm. Cu, 70 cm. distance) through 6 portals centered on the tumor, 450 r per treament, the two anterior fields being treated once and the posterior and posterolateral fields being treated twice. The tumor dose was not over 2,000 r.

The resectability rate in those receiving radiation therapy was lower than in those not receiving it, probably because of selection. The five year survival rates in those cases in which the tumor was resectable were the same in the two groups (about 50 per cent). The most significant difference in the two groups was in those cases in which the specimens showed involved lymph nodes. Those receiving roentgen therapy showed a 37 per cent five year survival; those without such therapy showed a 23 per cent five year survival. The group receiving roentgen therapy also showed a higher incidence of liver metastases, particularly as the time interval between irradiation and surgery increased beyond one month.

On the basis of this review, preoperative irradiation of carcinoma of the rectum was resumed at the Memorial Center.—*Henry J. Klos, M.D.*

Fletcher, Gilbert H., and White, Edgar C. Possibilities of supervoltage roentgentherapy in the management of cancer of the breast. *South. M. J.*, July, 1959, *52*, 805–812. (From: The Departments of Radiology and Surgery, The University of Texas, and the M. D. Anderson Hospital and Tumor Institute, Houston, Tex.)

The authors were exploring the possibility of extending the usefulness of radiation in the control of breast cancer by using supervoltage roentgen therapy and high energy teletherapy combined with orthovoltage (250 kv.) roentgen therapy.

The skin sparing effect and the increased depth doses achieved with supervoltage therapy alone are

conceded to be disadvantages in treating this disease On the other hand, orthovoltage therapy can reach cancerocidal doses in only a limited number of cases. Orthovoltage therapy was also associated with increased postoperative sloughing in the authors' cases which received preoperative roentgen therapy, if the breast was questionably resectable.

To gain the advantages of both types of radiation therapy, from 25 to 50 per cent of the tumor dose is given with 250 kv. and the remainder with cobalt 60 teletherapy. In general, the tumor doses reached in four weeks are: axilla, 4,000–5,000 r; supraclavicular and internal mammary lymph nodes, 4,000 r; mid-chest wall, 4,500–5,000 r. The skin reactions are less marked than with 250 kv. therapy alone and the doses are increased by from 1,000 to 1,500 r.—*Henry J. Klos, M.D.*

Gil y Gil, Carlos. Las siembras latentes en los tumores de la glandula mamaria. (Latent metastatic seedlings in tumors of the breast.) *Acta iber. radiol. cancerol.*, July–Sept., 1958, *13*, 247–258.

Roentgenographically and, sometimes, surgically demonstrable sternal metastases are dexcribed in 5 patients who underwent more or less radical mastectomy for carcinoma of the breast. Two of these 5 patients also received prophylactic roentgen therapy to the respective axilla. The sternal metastases were treated with local application of radium, to a reported dose of from 3,000 to 7,500 r.

Recalcification of the sternum was subsequently found on follow-up roentgenograms. Three of the 5 patients succumbed to generalized carcinomatosis, but only after prolonged survival (up to ten years from the date of the original diagnosis), and 2 patients were free of subjective or objective evidence of malignancy at the time of writing.

Theoretical considerations, with reference to the vascular connections of Batson's venous network, are presented to explain the alleged frequency of the usually latent sternal metastases from carcinoma of the breast. Even though based on conjecture, it would be well to remember this possibility and to ask for roentgen studies of the sternum in the course of a complete evaluation of a patient prior to making surgical decisions.—*E. R. N. Grigg, M.D.*

Hockin, J. G., and Hankins, F. D. Plastic and nylon applicators for cobalt treatment of cancer of the uterine cervix by the "revised" Manchester system. *Radiology*, May, 1959, *72*, 757–758. (Address: Suite 603, Medico-Dental Bldg., San Diego 1, Calif.)

A plastic and nylon modification for the application of cobalt in the treatment of cancer of the cervix by the "revised" Manchester system is described. The applicators are essentially the same as those designed for radium. Each unit of cobalt has an actual length of 14 mm. The strength is about 1.43 mc, which is equivalent to 2.5 mg. of radium. The ovoids are fitted with screw caps so that the cobalt can be permanently mounted in the plastic containers.—*A. W. Sommer, M.D.*

Wootton, Peter, and Cantril, Simeon T. Comparison of the use of standard depth dose data at 250 Kvp and 2 Mev by direct measurement of tumor exposure dose *in vivo*. *Radiology*, May, 1959, *72*, 726–734. (Address: P. Wootton, 1211 Marion St., Seattle 4, Wash.)

Conventional depth dose tables represent the distribution of exposure dose along the central axis of a roentgen-ray beam in a homogeneous, unit density phantom of effectively infinite dimensions. Since clinical material rarely conforms to such dimensions, corrections of depth dose data have been obtained by measurements after reducing the size of the phantom and by measurements of tumor dose either *in vivo* or in cadavers. The latter procedure has yielded valuable information which provides corrections for inhomogeneities, such as bone, and allows calculations of tumor doses from standard depth dose tables. Unfortunately, much of the work has been limited to the 200 kv. peak and 700 kv. peak range.

The authors have extended the investigation of *in vivo* measurements to include the 250 kv. peak (half value layer 2 mm. Cu) range in deep therapy and 2 mev. (half value layer 12.5 mm. Cu) in the supervoltage range. To permit such measurements the authors designed and developed a cold-sterilizable, intracavity ionization chamber for which the design and construction details are presented. Correct alignment of the field with the tumor site and the ionization chamber was achieved by conventional roentgenography and fluoroscopy and verified by exposure dose readings for successively smaller fields.

The tumor areas investigated in this study include those of the esophagus, nasopharynx, ethmoids, maxillary sinuses, tonsillar fossae, urinary bladder, tongue and the floor of the mouth, and rectum. The comparative differences between measured tumor dose and standard depth dose values are presented for each treatment area. In general, no correction was required for 2 mev. therapy, unless air cavities were traversed, while errors up to 25 per cent were common in fields treated by 250 kv. peak traversing bone.—*Edward B. Best, M.D.*